

THE BRITISH JOURNAL OF CHILDREN'S DISEASES.

FOUNDED BY GEORGE CARPENTER, M.D.

EDITED BY J. D. ROLLESTON, M.D.

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Original Articles.

PRE-NATAL HYGIENE AND PROBLEMS OF MATERNITY AND
CHILD WELFARE.*

By W. M. FELDMAN, M.D.,

Author of 'The Principles of Ante-natal and Post-natal Physiology.'

PRE-NUPTIAL HEALTH.

THE first and probably the most important point in connection with pre-natal hygiene is to choose one's parents. We know, of course, that there are many conditions which are transmitted from parent to offspring, *either* in that subtle manner which we call heredity, *or* as the result of certain pathological changes taking place inside the germ-cells of one or other parent, who may be suffering from certain microbic diseases or intoxications. We may take mental disease, epilepsy, certain forms of deaf-mutism, glaucoma, Friedreich's disease, albinism, hæmophilia, etc., as examples of true hereditary transmission, and syphilis as well as probably the effects of parental alcoholism on the offspring as instances of the second of the above modes of transmission. In all these cases a pre-nuptial health certificate on the part of each of the prospective partners would be a document of great eugenic value.

In this connection several problems suggest themselves. One is the question of sterilisation or segregation of those unfit to become parents; another is the question of consanguinity of marriage. As regards the

* Substance of a lecture delivered at the Royal Institute of Public Health on November the 1st, 1922 (Sir Frederick Mott, M.D., F.R.S., in the Chair).

first, my opinion is that segregation is both uneconomical and unphysiological and therefore unjustifiable, and as for sterilisation, the subject is too controversial to be discussed here. As regards consanguinity, I think I am right in saying that such marriages should only be discouraged if there is any undesirable hereditary trait lying latent or dormant in each of the intending partners. For instance, supposing two brothers, A and B, come from a family in which there is a tendency to mental disease, then it would not be wise for the children of A and B to intermarry. But supposing the family to which A and B belong to be perfectly healthy, but that, say, A's wife comes from a mentally unsound stock, then there would be no more risk for the children of A and B to intermarry than for them to marry outside the family. Moreover, if there is any very desirable trait in the family to which A and B belong, then it would be a eugenically most desirable thing for the children of these two brothers to intermarry with the object of perpetuating that valuable character.

THE GERMINAL OR RACIAL POISONS.

Syphilis.

Till very recently syphilis was generally believed to be the commonest cause of ante-natal death, but the most valuable and learned report of Dr. Eardley Holland to the Ministry of Health published early this year has deposed this disease from that unenviable position, and has placed it second on the list of causes of ante-natal mortality with a percentage figure of 16-20. Now, if we bear in mind the fact that there are close upon 140,000 annual ante-natal deaths in England and Wales, you will realise that 28,000 foetal deaths occur annually in England and Wales as the result of a cause which could have been prevented or ante-natally treated.

So much for the actual ascertained fundamental fact about syphilis and ante-natal life. There are, however, in addition several problems in connection with the subject which may appear to be of academic interest, but whose elucidation would lead to useful results in ante-natal and early post-natal hygiene.

Problems.—(1) Why do some expectant mothers who give birth to syphilitic infants give negative Wassermann reactions throughout pregnancy?

(2) Why should women who give birth to syphilitic infants have lucid intervals during which they may bear apparently healthy infants and then give birth to one or more syphilitic infants again?

(3) Why should Colles's law hold universally true, viz. that a syphilitic

infant will infect a hired wet-nurse but will never infect its own mother, even if the latter shows no clinical signs of syphilis and her blood gives a negative Wassermann reaction throughout her pregnancy?

The explanation offered by McDonagh and Amand Routh is that *Spirochæta pallida*, which is, of course, universally accepted as the cause of syphilis, may exist in the form of granules which McDonagh believes to be "spores" or the embryonic stage of the organism, but which others believe to be derived from the breaking up of the spirochæte. Be that as it may, it is suggested that these granules are carried by the head of the spermatozoon (which is too small to carry the spirochæte itself), and enter the ovum in the process of fertilisation. The ovum, therefore, becomes infected, although the mother may escape direct infection from her husband. The granules themselves are believed to be biologically inert but become infective after their development into the mature spirochæte—as they do after a certain variable period. The indirect infection of the mother from the foetus is believed to be prevented by the protective action of the ferments of the chorionic villi, which disintegrate the spirochæte in their attempt to pass into the maternal circulation.

On the other hand, in the cases where the woman bearing a syphilitic infant gives a positive Wassermann reaction the mother has been directly infected by her husband either before or during or after conception.

It has been found that in these latter cases spirochætes can be detected both in the maternal as well as in the foetal portions of the placenta, and if the chorionic ferment theory be true in the case of the Wassermann-negative expectant mother, it ought to be found that in such cases the mature spirochæte should be present in the foetal but not in the maternal portion of the placenta. This is a point which, as far as I know, has not yet been confirmed.

As regards Colles's law the explanation is easy in the Wassermann-positive cases, because in these cases the mothers have already been infected directly either before or during pregnancy and cannot therefore be reinfected. In the case of the Wassermann-negative mothers Routh believes that the spirochæte granules are present in the maternal portion of the placenta. After the birth of the child these granules develop into mature spirochætes—because the protective influence of the chorionic ferments has been removed—and thus infect the mother.

The criticisms which I have to offer on this very ingenious theory are:

(1) The existence of a granular stage in the life-history of the spirochæte is denied by many of the ablest bacteriologists, who believe the granules to be pure artefacts in the process of staining by Levaditi's method.

(2) There is no experimental evidence whatever to show that chorionic ferments have a destructive action on *Spirochaeta pallida* outside the body.

(3) Abderhalden has shown that the chorionic ferments do not disappear from the maternal tissues till fifteen days after labour, during which time it should therefore be possible for the mother to become directly infected by her infant.

This, therefore, is a subject upon which a great amount of useful research is most desirable.

The whole subject of pre-natal syphilis bristles with difficulties, for in addition to the problems I have just enumerated, there is the further fact that a positive Wassermann reaction in pregnant women has not necessarily the same significance as a similar result at any other time, since it has been shown that, at any rate during the early stages of pregnancy, there is an increased amount of lipoids (to which the Wassermann reaction is due) in the woman's blood.

Moreover, a negative Wassermann reaction in a man with a past history of syphilis is no definite guarantee that his offspring will be immune, although the wife may apparently escape infection. Hence not only are we faced with the old problem again, "When is a person with a past history of syphilis free to marry?" but there is the further problem, "How are we to deal with the unborn offspring of such a father?" Are we or are we not to apply antisiphilitic treatment to the foetus in such a case? I know of a case where administration of an arseno-benzol preparation to an expectant mother with a positive Wassermann reaction resulted in death of the mother, and post-mortem no evidence of syphilis was detected. Again I have had a case very recently where an expectant mother, six months pregnant, showed typical condylomata ani and vulvæ which in all reasonable probability were syphilitic, and yet both her own and her husband's blood gave negative Wassermann reactions. How is one to act here in the best interests of the foetus?

In spite, however, of these difficulties the fundamental fact remains that ante-natal syphilis can in most cases be diagnosed and treated and a large number of foetal lives saved by proper supervision and treatment of the expectant mother.

Ballantyne, who has done more for ante-natal pathology and hygiene than any other person in this country, has, in a paper read at the British Medical Association meeting in Glasgow this year, given the following noteworthy statistics:

Amongst expectant mothers who suffered from syphilis there were 606 stillbirths per 1000 in those untreated and only 50·7 per 1000 in those supervised and treated.

Alcohol.

Whilst there are no two opinions with regard to the harmful effects of the *Spirochæta pallida* upon the life and welfare of the foetus, opinions are not altogether unanimous respecting the influence of alcohol on ante-natal life. According to most observers alcohol is as much a germinal or racial poison as syphilis, whilst others believe that the effects of parental alcoholism upon the offspring are negligible. The question is not a new one. In the time of the Talmud, *i. e.* nearly 2000 years ago, it was remarked by a certain Jewish philosopher that it was only the daughters of alcoholic parents who require the aid of artificial means such as cosmetics and massage, etc., for the purpose of beautifying themselves; there is no such need on the part of the daughters of abstainers! It was also believed at that time that conceptions occurring during states of inebriety result in mentally-deficient offspring.

Of recent years the subject has been investigated experimentally, pathologically and statistically in both animals and human beings. Féré, by injecting various alcohols into hen's eggs at different stages of incubation, found that he was able either to stop development altogether or misdirect it in such a way as to produce monstrosities. He further showed that these deleterious effects varied in intensity with the toxicity of the alcohol.* Similar results have been obtained by Raymond Pearl in his experiments on birds which he exposed to alcohol fumes. He found that whilst his controls produced about 23 per cent. of infertile eggs, the proportion of such eggs produced by his experimental fowls was 60 per cent. Stockard and Laitinen, experimenting with mammals, found a large proportion of abortions, stillbirths, malformations and nervous disease in the offspring, and Nicloux, experimenting on women as well as on animals, found that when mothers were given a strong dose of alcohol about an hour before delivery, analysis of the blood taken from the umbilical cord of the infant immediately it was born showed the presence of alcohol within it, proving that the drug passes from the maternal into the foetal circulation.

Pathological.—Bertholet by post-mortem examinations on human beings, and Kyrle and Schoffer by experimenting on dogs, demonstrated degenerative changes in the sexual glands of alcoholic subjects.

Statistical.—Ballantyne found a high correlation between anencephaly and a family history of alcoholism, whilst Bezzola and others have produced statistics to show that the products of conceptions occurring during times of inebriety, such as carnivals, etc., resulted in a large percentage of stillbirths and idiocy. On the other hand, there are some who maintain that the statistical evidence has no real significance, and

* See table on p. 112 of my 'Child Physiology.'

that the experimental findings in animals cannot be truthfully applied to man, because the amount of alcohol to which the experimental animals have been exposed is relatively greater than is ever taken by human beings. Thus Prof. Karl Pearson and Miss Elderton in the 'Eugenic Laboratory Memoirs,' no. xiii, showed that Bezzola's statistics are mathematically unsound, and claim that their own findings as published in their 'Memoir' no. x to the effect that there is no appreciable difference between the offspring of sober and intemperate parents are true. Also Prof. Stockard could not convince himself that alcohol has ever caused abnormal development resulting in deformities in the human embryo, although he admits that the offspring of alcoholic parents show a greater number of stillbirths and general debility as well as a large proportion of sterility. Stockard, indeed, maintains that parental alcoholism, by killing off the weaker ova in the various stages of their development, eliminates the unfit specimens and thus ultimately benefits the race. Such a contention, however, cannot be treated seriously from the point of view of pre-natal hygiene, because on the same line of argument we ought to shut up our general hospitals, demolish our isolation hospitals, destroy our sanatoria and encourage slums and overcrowding as well as everything that helps to increase general and infantile mortality. *Such a hygiene by elimination eliminates all hygiene!*

We may summarise what we have said about alcohol and ante-natal life as follows :

(1) The statistical evidence is of a contradictory nature—as indeed is the case with most statistics.

(2) The experimental and pathological evidence leaves no doubt that (a) alcohol can and does pass from the maternal into the foetal circulation ; (b) the alcohol when so transferred has a prejudicial effect upon the foetus.

Other Toxic Agents that pass through the Placenta.

In addition to syphilis and alcohol, it has been proved that morphine, nicotine and lead salts pass from the maternal into the foetal circulation, and have, therefore, an important bearing on pre-natal hygiene. Also the toxins of infectious diseases may pass from the mother to the foetus, even if the mother is herself protected by previous attacks or vaccination. Thus, according to Ballantyne, a pregnant woman who has smallpox may associate with cases of smallpox with impunity to herself, but with considerable risk to her unborn child. Conversely, vaccination of the mother may confer immunity upon the foetus. The passage of drugs such as mercury, salvarsan, etc., is important from the point of view of pre-natal therapeutics.

The Toxæmias of Pregnancy.

We come now to another important group of causes of foetal mortality, viz. the toxæmias of pregnancy, including eclampsia, pernicious vomiting, etc. The pathogenesis of these conditions is believed to be brought about by toxins coming from the disintegration of the uterine tissue, caused by ferments of the chorionic villi—ferments whose action is primarily intended for the purpose of facilitating the anchoring of the ovum at the placental site. These toxins, which are carried into the maternal circulation, are under normal conditions either destroyed by the liver or sufficiently neutralised by antibodies manufactured inside the mother's blood, and then eliminated by the kidneys, bowels and other excretory organs. Hence, if the liver or excretory organs are defective, a state of pregnancy toxæmia results with the death of the foetus by suffocation. Dental caries—which, by the way, is a frequent complication of pregnancy owing to the demand of the foetus for calcium—and pyorrhœa give rise to toxins which throw an extra burden on both the liver and kidneys, and hence great factors in the prevention of foetal death due to true toxæmias of pregnancy are attention to oral hygiene, correction of constipation and of renal deficiency by means of rest, diet, etc., and treatment of the condition as soon as diagnosed.

It is just in cases of this description that the provision of ante-natal wards in connection with all ante-natal clinics would be not only of immediate public health value, but in virtue of their affording legitimate opportunities for useful research would greatly increase our knowledge of this distressing complication of pregnancy, and thus help to eliminate all foetal and maternal deaths attributable to pregnancy toxæmias.

Nutrition through the Placenta.

Whilst on the subject of transference of substances from the maternal into the foetal circulation, it will be profitable to refer briefly to the passage of food material through the placenta. Considerations of an anatomical, physico-chemical and experimental nature make it quite certain that food substances never pass *directly* from the maternal into the foetal circulation. As to how such transference is effected is not altogether certain. (a) According to the *vitalistic* theory, the walls of the chorionic villi exercise a selective action upon the various blood contents, allowing them to pass into the foetal circulation in quantities required by the foetus at each stage of its development. (b) According to the *osmosis* theory all the substances pass from one circulation into the other in accordance with the laws of osmosis, *i. e.* from a higher to a lower concentration. The matter is not purely of academic interest, but has a considerable

practical importance from the point of view of contracted pelvis. If, for instance, the osmosis theory is correct, then it should be possible in cases of contracted pelvis so to regulate the mother's diet, that by diminishing the mineral concentration of her blood the size of the foetus and the softness of its bones could be so adjusted as to make it capable of passing through the maternal passages at full term without resorting to Cæsarian section or induction of premature labour. The following analyses of specimens of blood obtained simultaneously from the mother and from the umbilical cord, show that whilst in the case of certain substances their concentrations are the same in the two bloods, suggesting diffusion by osmosis, in the case of other substances the difference in concentration is great enough to support the vitalistic theory :

Nature of material.	Maternal blood.	Fœtal blood.	Remarks.
	Mgram. per 100 c.c.		
Amino-acid nitrogen	5.9	7.9	Against osmosis (difference = 33 per cent. <i>in favour of fœtus</i>).
Sugar	132	115	Difference = 13 per cent. <i>in favour of mother</i> ; probably osmosis.
Fats	1485	1085	Definitely against osmosis.
Non-protein nitrogen	25.2	24.9	Almost exact balance; = osmosis.
Excretory nitrogen	17	16.6	Excretory products from fœtus to mother pass by osmosis.
Mineral substances	713	741	Difference = 4 per cent. <i>in favour of fœtus</i> . Not decisive.
Immune bodies	Equal concentration		Osmosis.

The subject is one upon which a vast amount of research is still necessary, although it may be mentioned that during the war, when many German expectant mothers suffered from scurvy, osteo-malacia, etc., as the result of lack of minerals in their food, the number of infants born with similar conditions was small, suggesting that salts pass from the mother to the foetus by the selective action of the chorionic epithelium.

INTRA-NATAL DEATHS.

Eardley Holland has shown that tears of the dura mater are responsible for a very large percentage of intra-natal deaths, especially in cases of breech presentation. This is a most important discovery from the point of view of preservation of foetal life, for both experimental work on animals* as well as clinical experience in the case of infants born during twilight sleep has shown that a newborn infant can remain apnoëic for as long as twenty minutes without any serious consequences to itself owing to the

* *Ibid.*, p. 276.

reserve of oxygen in its tissues. Hence it is a mistake to expedite unduly the birth of an aftercoming head, because the risk of foetal suffocation from compression of the cord is very slight compared with that of intracranial injury. It further follows from Holland's findings that as far as possible breech presentation should be converted into vertex presentation before labour.

In this connection again research is needed to ascertain the amount of compression the foetal head can stand with impunity. I suggest that by attaching a dynamometer to midwifery forceps the amount of compression exerted in every case of forceps delivery could be measured and recorded.

SUMMARY.

(1) Pre-nuptial clinics should be established where persons of both sexes can get expert advice as regards their fitness to marry.

(2) No person knowingly suffering from syphilis should be allowed to marry, and breach of such a rule should be made a penal offence.

(3) Every person who has at any time exposed himself to the risk of contagion by venereal disease should be declared free from both syphilis and gonorrhœa before marriage.

(4) All cases of pre-natal syphilis should be thoroughly supervised and treated as soon as diagnosed.

(5) All expectant mothers should as far as possible refrain from alcohol (at any rate in excess) or excessive smoking throughout pregnancy.

(6) All expectant mothers should be protected from exposure to infectious disease or dangerous industries such as lead and phosphorus or tobacco factories.

(7) Attention to the teeth, bowels and kidney functions is of great importance during pregnancy.

(8) Careful watch should be kept of the relative size of foetus and maternal pelvis, and any abnormal presentation should be converted into a vertex before labour.

(9) Ante-natal wards or beds should be provided in connection with all ante-natal clinics for the purpose of treating abnormalities of pregnancy, as well as for the purpose of carrying out properly directed and well co-ordinated biochemical, pathological and statistical research.

THE SEVERE BLOOD DISEASES OF CHILDHOOD: A SERIES OF OBSERVATIONS FROM THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET.

By F. JOHN POYNTON, M.D., F.R.C.P.Lond.,
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PART III.—PURPURA.

ANALYSIS OF CASES.

THE group of purpuras with which our paper is concerned contains ten cases in which the causation was obscure. Such are sometimes called examples of idiopathic or primary purpura, and we have excluded all cases associated with infectious diseases or other obvious cause.

For convenience we have divided this group into three sections:

The first, or purpura hæmorrhagica, is the most severe, and is associated with internal and external hæmorrhages and high fever.

The second, "Henoch's purpura," is characterised by sudden and recurrent attacks of abdominal pain, which may be of great severity.

The third contains cases in which there are subcutaneous hæmorrhages with little or no constitutional disturbance.

We believe that no dividing line can be drawn between these sections, and that they merge the one into the other.

The ages of the cases varied from 8 months to 9½ years, and the great majority were over 5 years.

The onset of the purpura was always sudden, but the duration varied; some of the cases had repeated attacks and were for years never free of some hæmorrhage.

The condition was a rare one, only ten examples occurring in three years. Once more we found that males were more often affected than females (at the ratio of 4-1), but in such a small series little importance can be attached to these figures in the face of the much larger series quoted by Holt and Thursfield, in which females preponderated.

In the introduction we have alluded to the question of the rôle of the blood-platelets in purpura hæmorrhagica. In our second case, kindly investigated by Dr. Bedson, of the Lister Institute, the number of platelets was reduced to 60,000 per c.mm. as against a normal of some 300,000.

Interesting and important though this subject of the blood-platelets undoubtedly is, we recognise that a reduction may also occur in some cases of anæmia gravis from deficient formation of megakaryocytes, and also in some cases of leukanæmia.

It would appear also that the essential point in the purpura formation is not the diminution of blood-platelets, but some poison acting upon the vital capillary walls, damaging their integrity.

ANALYSIS OF CHIEF SYMPTOMS.

The purpura varied greatly in degree: sometimes there were spots no larger than a "flea-bite"; again there might be extensive areas, and free bruising. The distribution was mainly on the buttocks, legs and arms; less frequently on the body, neck and face.

Hæmorrhages from the bowel, kidneys and nose were frequent, and might be copious and persistent, particularly from the kidneys. The urine was often claret-coloured, and contained blood-cells, blood-casts and leucocytes. *Hæmorrhages* in the tongue, in the cheeks and from the gums only occurred in the severe cases.

Hæmorrhages also occurred beneath the conjunctiva, from the stomach and into the wall of the larynx.

The *subperitoneal hæmorrhages* and *hæmorrhages into the intestinal wall* produce such remarkable symptoms that they have since Henoch's writings always attracted great attention. Our experience has entirely borne out the evidence of other writers as to the great difficulty of making a diagnosis from an acute appendicitis or intussusception. In a considerable number of cases in literature this has only been made after an unfortunate exploration. Great attention in such cases must be directed to the history, to the presence of any petechiæ or hæmorrhages from the nose or elsewhere, and to the tongue, which in these cases may be quite clean. A definite tumour may be felt through the abdominal wall, just as is the case in some examples of scurvy in infancy.

In some cases *pain* and *swelling of the joints* have attracted attention, and had usually preceded an outbreak of purpura.

Pyrexia occurs in the severe cases, and to a lesser degree in some of the milder ones.

In two cases there was a remarkable periodicity and regularity in the order of the appearance of the symptoms.

In one case the first events were a vomit and rise of temperature to 100° F. and 24 hours later the first appearance of purpura.

These cases of purpura were rather more frequent in the autumnal months.

The blood-picture was, as a rule, the following :

A moderate fall in the red cell count to about 4,000,000 per c.mm., a moderate increase in the leucocyte count up to 20,000 per c.mm., the differential count remaining normal.

In the fatal cases a great fall occurred in the red count, with a leucopenia and a gradual disappearance of the polymorphonuclear leucocytes.

The blood-platelets fell in number. The coagulation period did not appear to alter.

In some cases the blood-serum appeared to hæmolyse washed normal corpuscles, but not the patient's red blood-cells.

We could throw no light upon the ætiology from the histories of our cases.

The prognosis in the simple cases was good, but the fulminating examples of purpura hæmorrhagica were both rapidly fatal. Those of the Henoch type showed the well-known tendency to recurrence, and dragged on for some years. Treatment was unsatisfactory, apart from rest (*vide* section on treatment).

Section I.—Purpura hæmorrhagica.

There were two examples, both of which we give in some detail.

CASE 1.—Boy, aged 9 years. Admitted to hospital under Dr. Poynton on July the 10th, 1920, for "bleeding from the gums."

For three weeks the boy had been very fretful and quiet. Ten days later petechiæ appeared on the skin all over the body, and later "bruises" developed. About the same time bleeding from the gums commenced, which was worse in the night than the day, and shortly before admission hæmatemesis and melæna occurred.

The eyesight was not affected, but he had become slightly deaf, and had lost all appetite.

He was the youngest of three children, and there was no event in the personal or family history which threw light on this illness.

Condition on admission.—A well-built clever boy, he was very pale and his body covered with purpuric patches of various sizes and in different stages. From the gums there was persistent oozing of blood.

The premolar teeth were much decayed and the hæmorrhage from the gums most obvious at these points. The tonsils were large and there were petechiæ on them and the pharynx. The liver extended $1\frac{1}{2}$ finger-breadths below the costal arch, but the spleen was not palpable. The lymphatic glands were not enlarged. The pulse-rate was 108 per minute. The heart was not affected, but a faint hæmic systolic murmur was audible.

The respiratory and nervous systems were not affected.

The temperature on admission was 99.5° F. and rose to 100.8° F.

The blood-count was as follows: Red blood-cells, 1,760,000 per c.mm.; coagulation time, $6\frac{1}{2}$ minutes; hæmoglobin, 18 per cent.; colour index, 0.5; white cells, 1080 per c.mm.

Differential white count: Polymorphonuclear cells, 46 per cent.; small lymphocytes, 46 per cent.; large lymphocytes, 8 per cent.

No abnormal cells were seen. The urine was free from blood.

The diagnosis of purpura hæmorrhagica appeared the most probable.

The treatment decided upon was the subcutaneous injection of hæmostatic serum 2 c.c. at a time, and 10 c.c. of normal horse-serum. Turpentine was given by the mouth

in 10-minim doses. Improvement followed, the oozing from the gums practically ceased, and the purpura began to fade, the patient looking generally better. The treatment was steadily persisted with for a week.

Although there was this definite improvement the temperature remained raised, sometimes reaching 100° F., and there was occasional vomiting. On the seventh day a few fresh spots of purpura appeared, and from that date the condition grew worse. The pallor increased, and there was an icteric colour added and he was paler and weaker. The blood of his father, who was a powerful man, had been tested soon after his son's admission and he was found to be a universal donor, and his son also belonged to the same blood group. Accordingly it was decided to transfuse from the parent.

On July the 23rd, 1920, Mr. Twistington Higgins injected intravenously into the right saphena half a pint of citrated blood.

At the commencement of the injection the boy became restless and distressed and gasped for breath, the pulse remaining steady though rapid. Later he became drowsy, but coughed at intervals, and shivered. He passed a bad night, the temperature rising to 104° F. and his pulse flagging. The oozing from the gums persisted. That morning an extreme hæmoglobinuria developed and persisted most of the day. He failed rapidly, air-hunger supervened, and he died on the 25th, the temperature rising to 105° F.

Necropsy.—The heart was pale and fatty, and there were subserous petechiæ.

There was blood in the stomach, and submucous hæmorrhages in the intestines.

The liver showed areas of red necrosis and some slight cellular infiltration round the portal canals. There was much fatty change. The kidneys showed subserous petechiæ. The bone-marrow was pale but not watery.

The other organs showed no changes of note.

The great interest in this case was the sequence of events at and after the transfusion.

Though both parent and son belonged to the same blood group, it was evident at the time of transfusion that the child was disturbed by the injection. This disturbance was not great, for he was under an anæsthetic, and it was realised that the boy was very ill. Nevertheless the restlessness and gasping for breath at the outset were distinctly alarming. Throughout the time he had been in the ward previous to transfusion there had been no sign of any blood in the urine, but it is clear that the effect of the transfusion was to produce a hæmoglobinæmia of great severity.

We could compare this unfortunate event with Porter Parkinson's experience in the case we have already quoted under the section upon anæmia gravis.

CASE 2.—Male infant, aged 1½ years. He was admitted for "bruises," blood in the urine and bleeding from the mouth under Dr. Poynton on March the 3rd, 1921.

There was an eleven days' history of these symptoms.

The family history threw no light on the condition.

The history of diet since birth was as follows: Breast fed for five months, glaxo for four months, Savory and Moore's food four months. During the last month has taken milk, potato, gravy, and bread and butter.

The illness began with a cough and bronchitis, from which the child was apparently recovering when, on February the 21st, red spots appeared on the cheeks, then on the legs and elbows. On the 28th bleeding from the mouth commenced, and on March the 2nd blood appeared in the urine. The child took food well, and appeared in other respects in fair health.

Present state.—March the 3rd: A plump child with extensive purpura on face, arms and legs. The gums showed some purple discoloration at the bases of the incisor teeth. There were no subperiosteal hæmorrhages, but the child cried when moved. The urine was deep red with blood. The pulse was rapid—140—but the heart showed no lesion, neither did the lungs.

The abdomen was natural, and the liver and spleen were not enlarged.

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The child was treated dietetically on the lines of a scurvy, and 2 c.c. of hæmostatic serum injected.

The blood in the urine diminished, but the temperature, which on admission was normal, rose gradually to 104° F. on the 7th, and the child was listless, weak, and showed increasing pallor and great illness.

Dr. Bedson, from the Lister Institute, kindly undertook a blood-platelet count and reported as follows: (1) Bleeding time increased; (2) platelets, 60,000, as against the normal 300,000; (3) blood-platelet ratio, 53·5 as against 9·5. He considered it a case of purpura.

On March the 8th the child became much worse, and large blue areas appeared along the whole length of the spine. He appeared by his pallor and restlessness to be bleeding internally, and died that morning.

A partial necropsy only was permitted, and, except for numerous subserous petechiæ, nothing was discovered throwing light on the condition.

In these cases the question of scurvy is apt to arise, and particularly where, as in the second case, the patient is an infant. A careful inquiry into the history and the extensive subcutaneous hæmorrhages, with the diminished platelet account, served to distinguish the nature of the illness from scurvy.

An Example of Henoch's Purpura.

Boy, aged 7½ years, admitted under Dr. Robert Hutchison in June, 1920.

Had previously had measles and whooping-cough.

The complaint was bruising and purpura for the past nine months. On admission he was a healthy-looking child; small, petechial hæmorrhages were present over the trunk and extremities. He tended to bruise very easily. There was slight oozing from the gums. At fortnightly intervals this child vomited, his temperature rose slightly, and the following morning much purpura appeared on the body, arms and legs. During one of these attacks he had severe abdominal pain, a rise of temperature to 102° F., a rapid pulse tenderness in the right iliac fossa on palpation. In this phase the case might easily have been mistaken for one of acute appendicitis had he not been known to be a case of Henoch's purpura. This attack subsided in ten days. At his next attack a hæmorrhage occurred into the wall of his larynx, causing acute dyspnœa. He was discharged, having been in hospital for nine months, during which time these attacks continued to occur at fairly regular intervals. The blood picture showed no characteristic change. His serum seemed slightly hæmolytic to normal washed corpuscles. Seen a year later, he still showed many purpuric areas, and tended to bleed from the gums. He also had recurrences of abdominal pain.

An Example of a Simple Purpura.

Boy, aged 9½ years. Admitted under Dr. Hutchison in September, 1920.

When five years old he had whooping-cough; at six years hæmaturia, at seven measles. Six months previously he had passed blood in his urine. No purpura was noticed. A week ago purpuric spots the size of a half-crown appeared on the legs and arms, especially over the buttocks. His urine was bright red.

On admission large purpuric patches were noted on forearms, legs, and buttocks, but no blood was present in urine. The coagulation time of the blood was normal. The fragility was not increased. There was no pyrexia. The respiration and circulatory system were normal. The purpuric areas rapidly faded, and he was discharged cured in a month.

TREATMENT.

Advances in the treatment of severe anæmias in childhood are slow, and this series of cases shows clearly enough that the leukæmias when they come under our care are uninfluenced by any of our methods, and usually die rapidly.

Arsenic seems at this early age to be less effectual in producing temporary improvement than in the adult, and one experience of X-ray applications was not encouraging.

Benzene was ineffectual.

The only light at present is provided by those cases in which remarkable though temporary improvement occurs, but when we inquire into the circumstances that accompanied this improvement we find that it has not been the result of any special measures, but, on the contrary, supervened when no active attempts at treatment have been made. The fact itself, however, shows that there must be some natural effort at cure, and this attempt by Nature must have some explanation.

The value of *rest* in all forms of anæmia was apparent, and in the severe cases which eventually recovered it was obvious that premature exertion retarded their progress and recovery.

Warmth is another factor. We found open-air treatment in the colder months did not suit these cases, although when they were fairly on the road to recovery we had hoped this treatment with proper precautions would have been of value. It would appear that any strain on the resources of these patients, which in other convalescents might prove stimulating, caused a disproportionate injury to the blood.

A search for a *local cause* is as necessary in the child as in the adult, but it has been our experience in the worst cases to find very little assistance from such an inquiry. The occurrence of respiratory affections antecedent to a considerable number of the most severe cases did not lead to the discovery of any therapeutic indications, nor did active treatment of the attacks of sore throat which occasionally developed in the cases of leukæmia appear to influence the course of the disease.

Diet was of value in chlorosis and secondary anæmia, but of no avail in the primary anæmias.

One of the most striking observations upon the results of treatment that came under our notice was the danger of blood transfusion in anæmia gravis and purpura hæmorrhagica—an experience also met with by Porter Parkinson in his case quoted above. Although the usual precautions were taken as to the blood grouping of the donor and recipient, there resulted active hæmolysis with the most urgent symptoms. It would seem that in these patients there is some blood fault not to be

discovered in our present state of knowledge until the transfusion is done—and then it is too late. We have abandoned this procedure in anæmia gravis, leukanæmia and purpura hæmorrhagica.

Intestinal disinfectants were ineffectual in the grave anæmias, as was also hæmostatic serum.

Antimony tartrate for lymphadenoma, though in one case seeming to do good, was, in the next, a complete failure.

There is, however, a brighter side to the picture than this, when we come to other forms of anæmia.

Among the cases of acholuric jaundice one child who had failed to react to medicinal treatment and was steadily losing ground made a good recovery after splenectomy, and has up to the present remained in good health.

Cases of rickets with severe anæmia responded well to antirachitic and hæmatinic treatment.

Similarly cases of von Jaksch's disease with rachitic symptoms could be much improved, and with patience and care these cases slowly recovered unless of exceptional severity.

Chlorosis also responded in childhood to persistent treatment by rest, diet and iron, but in severe cases the treatment took many months to complete the recovery, and if this was stopped prematurely there was relapse.

If syphilis is responsible, a mixture of perchloride of mercury, perchloride of arsenic and perchloride of iron will be very helpful if it is well borne.

The removal of septic foci in the tonsils, gums and elsewhere will certainly assist the recovery from secondary anæmias.

It is clear that our failure in the treatment of leukæmia was dependent upon our ignorance of the essential nature of this disease. We do not yet understand the meaning of the profound changes in the blood in this disease, and are at a loss as to what lines to adopt either to prevent its development or to arrest its course.

It was also clear that in anæmia gravis we were defeated by the total lack of reaction to all measures in the worst cases, but the exact diagnosis is so difficult that in every case there is encouragement to leave no stone unturned to combat this form of anæmia, as our third case clearly proved.

THE PEL-EBSTEIN TYPE OF HODGKIN'S DISEASE.

By EDMUND CAUTLEY, M.D.Cantab., F.R.C.P.Lond.,

Senior Physician to the Metropolitan Hospital and the Belgrave Hospital for Children.

IRREGULAR pyrexia is common in Hodgkin's disease. The late Sir F. Taylor ('Guy's Hosp. Rep.,' 1906, lx, p. i) mentions various types of fever in this affection, viz.—(1) continuous, with slight diurnal variations; (2) alternating periods of fever and normal temperature; (3) daily variations of temperature in excess of normal limits; (4) mixed types in which at different times the above variations occurred. Pel and Ebstein, in 1887, independently described the recurrent or relapsing type—obviously only one variety of the fever which occurs in these cases. It is infrequent at all ages and decidedly rare in children. MacNalty has reported a case at 3 months and another at 8 years of age. Murchison's patient was a girl, aged 6 years.

The case now reported is that of a girl, aged 8 years on admission to the Metropolitan Hospital, where she remained under my observation until her death six months later. Strictly speaking, it should be classified under Taylor's fourth group, but the recurrent relapses in the later period of her illness were definitely of the Pel-Ebstein type. The peculiar grey-yellow or icteroid tint of the skin, first described by Pel, was very well marked.

The characteristic features of the illness can be summed up as follows: A prolonged local stage of glandular hyperplasia in the neck and a generalised stage of pyrexia, which assumed a relapsing type, severe and progressive anæmia, a yellowish-grey tint of the skin, enlargement of the cervical glands, liver and spleen, slight bronchitic attacks with breathlessness, sometimes semi-delirium, erythropenia and leucopenia, and a fatal issue. There was no evidence of tuberculosis—an alternative diagnosis—and the Pirquet reaction was negative. Unfortunately no post-mortem examination was obtained.

For a period of three weeks the fever was of the common irregular type, maximum 102·4° F. During the next week it kept fairly well at about the normal level, and in the next eight weeks there were roughly alternating weekly periods of higher and lower temperatures. From January onward the relapses took the more definite form of a fortnightly periodicity, though not absolutely regular. Each such attack of fever resembled the common type of relapse in an enteric fever and lasted about a fortnight. The 12-hourly chart showed a step-like rise of temperature for 4 or 5

days, a period of maximum temperature for 4 to 7 days, and a fall by lysis. Some of the relapses were preceded by apyrexia for about a week. From March the 3rd to April the 2nd the attack of fever was more irregular and prolonged, and suggestive of an intercurrent relapse. The final bout followed 12 days' apyrexia and showed the step-like rise in its most marked form. It lasted two weeks, and was subsiding by lysis in the usual way when the child succumbed from exhaustion. So suggestive was the course of the temperature of one of the enteric groups of fevers that it was deemed necessary to try agglutination tests in order to exclude the possibility of such a causation; the results proved absolutely negative.

CLINICAL ABSTRACT.

Ivy R—, aged 8 years, was admitted to the Metropolitan Hospital on October the 14th, 1921, and died on April the 28th, 1922. She was the youngest of eight children, all of whom were said to be delicate and have weak chests. The father was stated to be phthisical. According to the mother, the child had always been delicate and under medical advice for enlarged glands in the neck. In 1916 she had diphtheria and was in hospital for nine months. In 1918 the glands became much larger, and open-air treatment was recommended by a London surgeon. After this they varied in size from time to time. In January, 1921, she went to the Margate Sea-Bathing Infirmary, and in February a gland was removed, which was said to be lymphadenomatous. Although getting weaker, she remained there until June, and had lived an invalid life since. On the advice of Mr. Homi she was brought to the hospital. Her chief symptoms have been variable appetite, frequent nausea, constipation, increasing anæmia, abdominal enlargement and loss of weight, and cough for 14 days.

Condition on admission.—Good nutrition, marked anæmia and a yellowish-grey tint of the skin; clean tongue and good teeth, no pyorrhœa; enlarged glands in both sides of the neck, especially on the right side, which shows the scar of the operation. No adventitious sounds in the chest, save hæmic murmurs, and no evidence of enlarged thoracic glands; a somewhat distended abdomen with moderate enlargement of the liver and spleen.

Course.—Although under observation for a long period, there is comparatively little to note in reference to the symptoms. The chief features of the case were the progressive anæmia, the pyrexia and the blood-counts. The glands in the left side of the neck became quite small soon after admission, and did not again increase in size. Those on the

right side became smaller, but they enlarged from time to time, though not always with the pyrexial attacks. The spleen was never very large. On October the 21st it extended $1\frac{1}{2}$ in. below the costal margin, and it was both larger and harder ten days later. After this it became smaller. On January the 8th there was a sudden attack of pain in the splenic region, with tenderness and friction, but no apparent increase in size. The pain continued more or less for ten days, and was probably due to thrombotic infarction. On January the 27th a relapse was in full blast, but there was no increase discoverable in the size of the glands or spleen. During February there was subconjunctival hæmorrhage in the right eye, and in March an attack of herpes labialis and occasional slight epistaxis. The final attack of fever began on April the 13th, and the glands became larger, though not as big as on admission. Epistaxis was severe on April the 25th, and a rigor occurred on April the 28th shortly before death.

Beyond these signs there was nothing to note save the increasing anæmia, the tint of the skin, slight bronchitic attacks, with a few crepitations in the lungs, and some delirium during two of the febrile relapses. The liver did not increase in size. Throughout the illness the child's appetite was good, sometimes enormous, except at intervals. Even during pyrexial stages it was occasionally excessive, though it generally failed at these periods. The weight varied comparatively little. On admission it was 40 lbs., and between November the 25th and March the 14th it varied between 42 and 43 lbs. On March the 21st, the last weighing, it was $41\frac{1}{2}$ lbs.

For the reports on the blood I am indebted to Dr. J. Andrew, Pathologist to the hospital. No growth was obtained by culture.

Blood-Counts.

Date.	Red cells.	Hb per cent.	C.I.	White cells.	Poly-morphs.	Lymphocytes.	Large mono-nuclears.	Eosino-phils.	Baso-phils.
1921.									
Nov. 17 .	1,600,000	29	0·8	5000	3125	1700	100	62	13
Dec. 9 .	1,750,000	25	0·7	4000	2210	1640	120	20	10
„ 30 .	1,250,000	26	1·0	4000	2320	1480	140	20	40
1922.									
Jan. 10 .	1,400,000	20	0·7	2500	1513	862	112	13	0
Feb. 1 .	1,100,000	13	0·6	2000	600	1320	68	20	0
„ 17 .	—	—	—	2200	748	1364	88	0	0
April 3 .	1,270,000	18	0·7	3400	1751	1513	68	51	17
„ 15 .	1,000,000	15	0·7	1700	884	765	51	0	0

Relative Percentages of Polymorphs and Lymphocytes.

	Nov. 17, 1921.	Dec. 9.	Dec. 30.	Jan. 10, 1922.	Feb. 1.	Feb. 17.	April 3.	April 15.
Polymorphs	62.5	55.25	58.0	60.5	30.0	34.0	51.0	52.0
Lymphocytes	34.0	41.00	37.0	34.5	66.0	46.0	44.5	45.0

The obvious features are the profound and increasing anæmia and the leucopenia. The reduction of red cells to one million per c.mm. exceeds that in MacNalty's boy, 8 years old, in whom the number fell to $1\frac{1}{2}$ million and the hæmoglobin to 10 per cent. Even lower counts have been recorded in children. Dr. J. Porter Parkinson showed at the Children's Section of the Royal Society of Medicine in April, 1919, as a case of recovery from aplastic anæmia, a boy in whom the blood examination at one time had yielded only 580,000 red cells and 2600 white cells, of which 76 per cent. were lymphocytes (*vide* BRITISH JOURNAL OF CHILDREN'S DISEASES, 1919, xvi, p. 1).

It is noteworthy that the number of white cells fell progressively, except for the increase noted on April the 3rd, two days after a prolonged attack of fever. Possibly this was due to a final attempt at recovery. On February the 1st, at the end of an apyrexial period, on February the 17th, during the middle of a relapse, and again during the final attack, the number of polymorphs were greatly decreased, whereas the number of lymphocytes remained fairly constant. I cannot account for the relative differences noted on January the 10th, at the end of a course of sodium cacodylate injections, and during the attack of pain in the splenic region. No myelocytes or nucleated red cells were found, and the red cells present appeared normal.

GENERAL REMARKS.

Hodgkin's disease (lymphogranulomatosis maligna) was defined by Ziegler in 1911 as a specific granulomatous affection with a special predilection for the lymphatic tissues at first. There are various clinical types, described as acute, local, generalised, splenomegalic, like mediastinal tumour, and like typhoid. Undoubtedly there are two definite stages. The first stage is local and generally glandular, and varies in duration from weeks to years. In this child the glands had been enlarged for years. About half the cases begin in the cervical glands, and next in order of frequency in the axillary, supraclavicular and inguinal glands. About 10 per cent. begin in the spleen and a similar proportion in the mediastinal, retroperitoneal or other internal glands.

The stage of generalisation is characterised by fever, anæmia and cachexia, an enlarged spleen in two-thirds of the cases, localised, and sometimes wide-spreading glandular enlargement, with leucopenia and blood of the type of secondary anæmia. The other organs affected include the bone-marrow, kidney, liver, pancreas, and sometimes the skin and intestinal tract.

The fever is suggestive of an infective origin of the disease, but, so far, there is no reliable evidence thereof. In this patient no local focus of infection could be found, and blood cultures were negative. As a rule, the pyrexia is an added phenomenon to a local disorder, generally glandular. Parkes Weber regards it as indicative of a stage of dissemination or generalisation of the disease—a kind of “septicæmia of Hodgkin’s disease.” In view of the periods of apyrexia, sometimes prolonged, this is an unsatisfactory view. It is more reasonable to ascribe the pyrexial bouts to localised necrosis of lymphadenomatous tissue and toxic absorption. Weber found areas of centro-acinous necrosis in the liver and pancreas of his fatal case (‘Proc. Roy. Soc. Med.,’ 1916–1917, Clin. Sect., p. 42). The occurrence of the attack of pain in the splenic region during one febrile relapse in this child affords some support to this hypothesis, if it was due to local necrosis in the spleen. In other words, the fever is merely a sign of the secondary necrosis and the primary origin of the disease awaits discovery.

Treatment proved absolutely unavailing, though it may have prolonged life. Arsenic and reduced iron were given by mouth, sodium cacodylate and colossal manganese intramuscularly, antimonium tartrate, gr. $\frac{1}{4}$ in 2 c.c. normal saline intravenously, and hæmoplastin for the epistaxis. The greatest improvement in the child’s general condition followed the daily administration of an ounce of brandy.

CEREBRAL HÆMORRHAGE IN A NEWBORN CHILD.

By C. F. T. EAST, M.A., B.M., B.Ch.Oxon., M.R.C.P.Lond.

A MALE infant was born in the Labour Ward of King’s College Hospital at 12.55 a.m. on Saturday, July the 22nd, 1922. The mother was healthy and had had a normal pregnancy. She had had one other child three years before, now a healthy boy. The baby was full time, and labour was easy, lasting 2 hours 20 mins. The presentation was L.O.P., turning to L.O.A.; no instruments were used, nor was there any holding back

of the head during delivery. Weight at birth was 6 lb. 10 oz. Just after birth two bluish marks were noticed on the left shin.

On Saturday afternoon the mother remarked that the baby did not seem to take the breast very well, and the nurse thought that it had a peculiar cry. It had scratched its face, and the small mark bled very freely. During the evening it would not suck at all, and a blood-stained froth came up into its throat. Its temperature was then 96° F.

At about 7 p.m. it looked very grey. During the night it seemed very ill, and at about 6 a.m. it went a very bad colour, though afterwards it became markedly flushed.

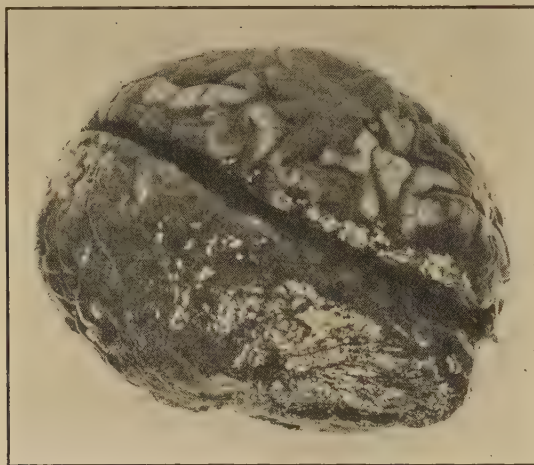


FIG. 1.—Upper surface of brain.

A hypodermic injection of strychnine was given about this time, and afterwards it went rigid, holding its head turned to the right.

At no time did it vomit, nor was there any actual fit or convulsion. Respirations were gasping, and it twitched its arms and legs at times.

There was a little oozing from the cord. Normal meconium was passed.

The fontanelle was now noticed to be tense. There were purpuric spots on both legs, and also on the backs of the arms and right scapula.

The respirations were gasping.

When seen about 11 a.m. on Sunday morning the baby was very white.

The temperature was 96° F., and it was drawing gasping breaths at intervals of about a quarter of a minute. The eyes were half closed and seemed rather bulging. It lay limp in the basket, making occasional jerky movements with its arms. The fontanelle, indeed the whole

skull, was extraordinarily tense, showing a very high intracranial pressure.

No pulse could be felt at the wrist or fontanelle, but the stethoscope showed it to be 164. The heart-sounds were faint, but normal. The breathing did not allow any signs to be made out in the lungs. Blood-stained froth was wiped from the pharynx. Blood was oozing slightly from a point just below the junction of the cord. Patches of purpura were on the shins (about 4 mm. in diameter) and on the back of the arms and shoulders; some of the spots were mere petechiæ. No knee-jerk was obtained.

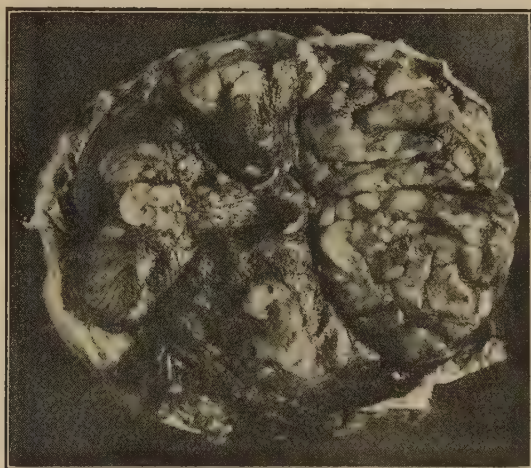


FIG. 2.—Lower surface of brain.

With the ophthalmoscope it was seen that the retinae were covered with hæmorrhages.

The baby died in about a quarter of an hour.

There is absolutely no history of any hæmorrhagic tendency in the family.

Autopsy.—Patches of purpura and petechiæ were seen as described above.

Both lungs were covered with scattered hæmorrhages, mostly about the size of a threepenny-bit. These hæmorrhages extended into the lung. There was nothing abnormal about the parietal pleura.

The pericardial sac contained a normal amount of fluid; at the base of the aorta and the pulmonary artery were two purpuric spots. Under the visceral pericardium there were a few scattered petechiæ; the parietal pericardium was normal. The thymus was normal.

The bronchi and trachea contained a little blood-stained froth.

The peritoneum showed no abnormality, and there was no blood in the intestines or stomach. The liver, spleen, kidneys and suprarenals were natural. There was no blood in the bladder.

The caput succedaneum was not abnormal, but some blood had effused under the pericranium of both parietal bones. On opening the dura mater a great deal of blood escaped; on removal blood and clots were seen all over the surface of the brain, extending everywhere round the base down into the spinal canal. The photographs give an idea of the extent of the hæmorrhage. No bleeding had taken place into the substance of the brain itself or into the ventricles. There was no injury to the meninges, and no source of the blood could be found.

It is unfortunate that there was not time to examine the blood before death.

A microscopical section of the lung showed that it was well expanded, and that in places the alveoli contained blood.

In this case one cannot easily explain all the hæmorrhages by trauma. Of course, there must have been some strain on the child during the birth process although labour was so easy. The ocular hæmorrhages may have been caused at this time. Koenigstein (4) found hæmorrhages round the optic disc in 10 per cent. of the cases he examined. Schlich (7) found them in 30 per cent., and Sicherer (8) in 20 per cent. The last observer states that they have no relation to hæmorrhagic disease, and traces a relation between the presentation and the eye affected. They occur more frequently in large children, and when forceps have been used. Von Reuss (6) agrees that the strain of the birth process is the cause.

Such extensive bleeding into the meninges of apparently spontaneous origin seems rare. Trauma, such as forceps delivery, is usually the cause, according to Spencer (9). Green and Swift (2), in a series of 51 cases found meningeal bleeding in 2 only, and they were not extensive. In a series of 32 cases Townsend (10) found none at all.

Bleeding into the lungs occurred in no case of these series, while Kilham (3) found small purpuric spots in the lungs in one case in ten.

Umbilical hæmorrhage is most common in these conditions. It occurred in each of Tuley's (11) 38 cases. Abt (1) has reported 13 cases of spontaneous hæmorrhage in the newborn, each of which showed purpuric skin lesions such as are described above.

One can hardly account for the bleeding in this case by trauma alone, unless there were present already a condition of the blood or capillaries which would predispose towards it.

The purpuric spots point to the presence of some such change.

The persistent bleeding from the small scratch on the face is similar to the free bleeding from a small wound in purpura. Larrabee (5) found a distinct family history in 37 out of 38 cases which he classes as hæmophilia in the newborn. Extensive inquiry has shown nothing in the family history here, though in the absence of the coagulation time one cannot exclude it. There is no positive evidence of infection; one can only class the case as one of hæmorrhage, due to changes in the blood or capillary walls, possibly as a result of infection or toxins. Very slight trauma may have been enough to cause bleeding under such conditions in some of the organs; in others it would appear to have been spontaneous.

My thanks are due to Dr. Hugh Playfair for his kind permission to publish this case, which occurred in his ward.

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Royal Society of Medicine.

SECTION FOR THE STUDY OF DISEASE IN CHILDREN.

Friday, March the 24th, 1922.

The President, SIR ROBERT JONES, K.B.E., in the Chair.

Specimen from a Case of Aneurysm of the Ductus Arteriosus.—Dr. ROBERT HUTCHISON (see BRITISH JOURNAL OF CHILDREN'S DISEASES, 1922, xix, p. 85).

Dermato-polyneuritis (Acrodynia; Erythrœdema).—Dr. HUGH THURSFIELD in collaboration with Dr. DONALD PATERSON (*ibid.*, p. 27).

Erythrœdema.—Dr. F. PARKES WEBER (*ibid.*, p. 17).

Case of Double Inguinal Hernia in which Both Sacs were removed through a Single Transverse Suprapubic Incision.—Mr. PHILIP TURNER showed a boy, aged 2 years and 2 months, who had been admitted to hospital for double inguinal hernia. At the operation a transverse incision, about 3 in. in length, was made 2 in. above the pubes. The sheath of the rectus was exposed, and the skin and superficial tissues were undercut, so that on retraction the aponeurosis of the external oblique was exposed on either side as far as Poupart's ligament. The left side, on which the hernia was the larger, was first dealt with. The method employed for the removal of the sac was that described by Mr. Turner in 1912 (see *BRITISH JOURNAL OF CHILDREN'S DISEASES*, 1912, ix, p. 123). An incision was made through the external oblique, over the internal abdominal ring, and the lower margin of the internal oblique was then drawn up so as to expose the sac and the spermatic cord just below the internal ring. After these structures had been freed and drawn through the incision in the external oblique, the sac was separated from the cord and isolated up to the internal ring. Below it was found to be continuous with the tunica vaginalis, so that after it had been ligatured above a second ligature was applied where it joined the tunica vaginalis and the intervening part removed. The testicle, which had been pulled up into the wound, was then pushed back along the inguinal canal into the scrotum, and the incision in the external oblique was closed. The superficial tissues were then retracted to the right, and a terminal sac of considerable size, but not continuous with the tunica vaginalis, was removed by the same procedure as that employed on the opposite side. The advantages of the operation were that the wound was well away from the groin, and that it was in a favourable position for subsequent dressings and nursing. The incision was particularly useful for those cases in which there was a definite hernia on one side, while on the other there was perhaps a slight swelling or a doubtful history of the occasional appearance of such a swelling. In that case the hernia could be treated and the other inguinal canal could be explored for the presence of a sac.

Case of Exophthalmic Goitre.—Dr. E. BELLINGHAM SMITH showed a girl, aged 13 years and 9 months, who had been taken to a throat hospital a year ago for some difficulty in swallowing. There was a swelling in the neck, and three months later exophthalmos developed. She now had exophthalmos, a large goitre and a pulse-rate of 120 to 140, was nervous and emotional, and had attacks of palpitation. There was a fine tremor of hands.

Specimens from Case of Obesity.—Dr. E. BELLINGHAM SMITH and Dr. R. DONALDSON related the subsequent history of the case shown at the last meeting (see *BRITISH JOURNAL OF CHILDREN'S DISEASES*, 1922, xix, p. 146). The patient was admitted to hospital to see if, like a diabetic, he would react to starvation diet; the result was that on the fifth day of this diet there was no reduction of the sugar, and there was a large increase in the diacetic acid and acetone. He was threatened with diabetic coma and became drowsy, so he was put back on full diet. He recovered for three or four days, then had convulsions and died.

Necropsy.—There was an incipient moustache and the hair had a feminine distribution. On the trunk, at the level of the umbilicus, the fat had a depth of $2\frac{1}{2}$ in. There were dilated veins and multiple small hæmorrhages

under the skin. The brain weighed 2 lb. and the pituitary gland appeared to be normal; its weight was 0.65 grm.; the pineal gland was small—weight 0.068 grm. No macroscopical lesion was discovered. No thymus could be found, but an enormous pad of fat covered both lungs. The aorta was hypoplastic. The suprarenals were slightly larger than normal, and their weights were respectively—right, 12.5 grm.; left, 13.5 grm., very soft and flabby. There were no cortical nodules. Near the hepatic flexure there was, in the transverse meso-colon, a small nodule, half of which had been exhibited. It was firm, and on section it was seen to consist mainly of blood. There was no yellow mottling. There were pale areas of degeneration and necrosis. The section shown was that of the cells that had been least damaged. Extending from the nodule upwards over the superior surface there was a quantity of brownish fluid, and adherent to the liver, greyish, putty-like material—evidently altered blood-clot. A similar but smaller nodule was found near the head of the pancreas, but not in it. The pancreas itself showed pale areas of necrosis, and in the fat round the pancreas were areas of fat necrosis. The testicles were infantile and weighed 2.5 grm. each. They showed considerable fibrosis, very few interstitial cells, and no evidence of spermatozoa formation. Dr. Bellingham Smith agreed with Dr. Parkes Weber that it was a case of hypernephroma growing from an accessory suprarenal.

Case of Hemiplegia.—Mr. B. WHITCHURCH HOWELL showed a boy, aged 11 years, who had had traumatic hemiplegia probably when he was born. His previous history was not available. When brought to Mr. Howell for right hemiplegia he showed a depressed fracture on the left side of the skull. He was intelligent, and appeared to have no fits. Re-education was first tried, and then the depressed fracture was removed in November, 1921. There had been distinct improvement since, but there still remained a definite hemiplegia of the right upper limb which required correction. Mr. Howell proposed to divide a certain portion of the fibres of the median nerve going to the pronator radii teres, and to explore the musculo-spiral nerve to see if he has apparent paresis of the extensors of the wrist and fingers. If so, instead of dividing some of the motor nerve-fibres going to the flexors of the fingers, Mr. Howell proposed to transplant one or more of the flexor group to the extensor aspect of the wrist and fingers, to give the boy the power to dorsiflex the wrist and fingers.

Case of Hydatid of Pleura and Lung in a Boy, aged 8 years.—Dr. R. C. JEWESBURY.—The patient had been sent to hospital with a history of having had a “chill” about five months ago, and afterwards he complained of pain in the back and chest. Two months ago he was seen by his doctor, who diagnosed “congestion of lungs” at left base; this was thought to be followed by empyema and he was sent to hospital. When first seen in hospital he appeared to be well nourished but rather pale. Respirations 28, pulse 92, temperature normal. There was a large area of dulness at the left base with some pleural friction in the upper part of the left axilla. The heart was slightly displaced to the right. Breath-sounds, vocal resonance and fremitus were diminished over the dull area. The note on percussion was impaired as were also the breath-sounds over a fairly large area at the right apex. The left chest was explored and a syringe-ful of a perfectly clear colourless fluid looking like water was with-

drawn. Blood examination: Red cells, 4,250,000; hæmoglobin, 64 per cent.; colour index, 0·8; white cells, 5680; polymorphs, 60 per cent.; eosinophiles, 6 per cent.; lymphocytes, 30 per cent.; large hyalines, 4 per cent. The left chest was then aspirated, and 200 c.c. of slightly opalescent fluid containing shreds of lymph-like material were drawn off. Examination of the fluid showed scolices of *Tænia echinococcus* in large numbers. Hydatid complement reaction weak positive; precipitation reaction weak positive. The eosinophiles increased from 6 to 10 per cent. after aspiration. Dr. Jewesbury said that hydatid disease, particularly of the lungs or pleura, was comparatively rare in children in this country. At St. Thomas's Hospital during the last fifteen years there had been in all 21 cases—7 in males and 14 in females; the ages varied from 19 to 60, with 1 female child aged 3½ years, who had a hydatid cyst of the liver. The cases were as follows: Hydatid in liver, 16 cases; hydatid in lung, 2 cases; hydatid in kidney, 1 case; hydatid in peritoneum, 1 case; hydatid in spinal cord, 1 case.

Generalised Muscular Hypertrophy in a Boy, aged 10 years.—

Dr. R. C. JEWESBURY.—The patient had been sent to hospital as a case of ? facial paralysis. He had always been an ailing child. He began to walk at two years and was very backward mentally. He had a difficulty in speaking, and speech was accompanied by spasm of the facial muscles. There was no facial paralysis. He was unable to protrude the tongue to the full extent. There was no evidence of disease in the central nervous system. He had always been of a lazy disposition and did not care for games at school. Family history: He was one of nine children, all the others being normal. There was an extraordinary condition of hypertrophy of all his muscles, but this was particularly marked in the muscles of the shoulders, arms, chest and back. When stripped he looked like a young prize-fighter. It appeared to be a true hypertrophy, for his strength was in proportion to his muscular development. His weight was 3 st. 11 lb., and his bones were normal for his size. There was no abnormality of sexual development. A skiagram of the base of the skull showed a perfectly normal sella turcica, and there were no eye changes.

SECTION OF DERMATOLOGY.

May the 18th, 1922.

Case of Fox-Fordyce Disease.—Dr. H. W. BARBER showed a girl with the extremely rare condition described first by Fox in 1902, since when other cases had been published by Fordyce, Brocq, Haase and others. Fordyce named it "chronic, itching, papular erythema of the axillæ and pubes," and was inclined to classify it with simple lichenification, or the "névrodermite chronique circonscrite" of the French. Dr. Barber did not think it was of this nature, one reason being that the distribution was always the same, namely in the axillæ, pubic region, and to some extent on the presternal skin. Another point against its being simple lichenification was that X rays, even in big doses, had no effect on the eruption. The patient had now less itching, but he attributed that rather to the improvement in her general health than to the influence of the rays. Probably the patient secreted

some irritant through the sweat-glands. Histological examination confirmed Fordyce's description but did not throw much light on the ætiology. The patient had had the eruption just over two years.

Case of ? Angioma Serpiginosum.—Dr. E. G. GRAHAM LITTLE showed a boy, aged 12 years, with a nævoid condition which had first appeared on the right leg in a linear distribution near the ankle at the age of $2\frac{1}{2}$ years, and had steadily progressed up the leg till it had reached the mid-thigh. The patches took the shape of punctate hæmorrhagic spots. The patient was healthy and there had been no family disease.

Case of Adenoma Sebaceum.—Dr. S. E. DORE showed a boy, aged 6 years, with adenoma sebaceum of the Pringle type. There was vascular dilatation accompanying the sebaceous growths. It was noticed on the cheek at the age of one year, and similar lesions rapidly followed on the opposite cheek and on the nose and chin. The lesions were grouped about the nose, labial folds, lower parts of the cheeks and chin, and were stated to be gradually increasing in number. They varied from a pin's point to a pin's head in size, and were very slightly raised above the surface of the skin, except in one instance, there being a more elevated and larger flat growth as large as a pea in the centre of the right cheek. There was no evidence of mental deficiency, but he had had fits, which began at three months of age and lasted until he was a year old. He also had a flat growth on the right side of his chest which Dr. Dore thought was fibromatous. Molluscous growths were commonly found about the iliac and lumbar region in these cases. Electrolysis was undoubtedly the best treatment, but in hospital practice this was difficult.

Bullous Ichthyosis.—Dr. W. J. O'DONOVAN showed a female infant, aged 1 year 11 months, born at full time. There is no record of any skin affection in the family. There were no spots on the child at birth, but on the second day crops of blisters appeared and had continued to do so at frequent irregular intervals. When Dr. O'Donovan first saw the patient, in September, 1921, only an impetiginous condition was noted. Under treatment the scales disappeared and thick patches of epidermis or bare areas of burst blisters were visible. Temporary improvement followed a week's rubbing with mercury ointment, then after a $\frac{1}{4}$ pastille dose of X rays all over the eruption cleared up entirely. A month later (on April 6th, 1922) the child was readmitted, now presenting a tylotic condition of its hands and feet and a marked hyperkeratosis over its elbows, knees and neck. Over the abdomen there were segmented-like bands of cross-hatched thickened epidermis. The differential blood-count was normal; the Wassermann reaction was normal; there was no adenopathy. An injection of milk produced a marked local reaction, but the complete exclusion of milk and milk products from the diet produced no alleviation.

On commenting on this case the PRESIDENT (Dr. H. G. ADAMSON) said he thought this was a case of linear nævus. These multiple streaks often appeared months, even years after birth, and sometimes they were not noticeable until five or six years of age. It seemed to him that the blisters occurred only upon the areas of linear nævus and not upon the rest of the skin. He did not regard it as a case of epidermolysis bullosa, but as a warty linear nævus with bullous formation at the site of the nævus.

SECTION OF ORTHOPÆDICS.

April the 4th, 1922.

Case of Arthroplasty of both Hips.—Mr. O. L. ADDISON showed a girl, aged 15 years, first seen in May, 1915, with ankylosis of both hips, numerous abscesses and sinuses which had followed an attack of "rheumatism" in November, 1914. Murphy arthroplasty right hip August, 1916. Leg put up in abduction three to four weeks. Free flexion to right angle and movement painless. Murphy arthroplasty left hip January, 1918. No voluntary movement after operation; a few degrees only under anæsthetic. Patient not seen between March, 1918, when no movement was present in left hip, until March the 30th, 1922, when X rays showed complete absorption of head and neck on right side and very small part of neck remaining on left side in acetabulum. Free flexion and good abduction on right side. Flexion to right angle of left with about 30° abduction. Never had any pain or trouble in walking. The case suggested that arthroplasty had no advantage over excision.

Case of Arthritis of the Hip in a Girl, aged 9 years.—Mr. B. WHITCHURCH HOWELL.—When 6 months old the patient had an abscess of the left hip-joint, which was drained. When seen in February, 1922, the left lower limb was 1 in. short, and there was abduction of the femur, with flexion deformity of 60°. The hip was stable with a fair range of movement. X rays showed destruction of the head with broadening of the upper part of the femur. The points raised were: (1) Should a subtrochanteric osteotomy be done? (2) Should the anterior superior iliac spine be detached, with extension? (3) Or should (1) and (2) be done?

May the 2nd, 1922.

Case of Renal Dwarfism.—Dr. DONALD PATERSON showed a boy, aged 6 years. History: When aged 2½ it was noticed that he was getting knock-kneed. Had progressively become more and more deformed since. Height, 35 in., the normal height for his age being 44 in. Interstitial nephritis present. Urine: Specific gravity 1004 to 1014; cloud of albumin always present. Blood-pressure, 100 mm. Hg. Urea concentration test: Maximum output, 1.9 per cent.; normal, 3 to 4 per cent. Eyes normal. X-ray examination: (1) "Woolly" appearance of diaphyses at wrists; (2) fracture and irregularity of epiphysal line at lower end of femur. It was suggested that the agent which brought about the renal fibrosis also caused the bony changes. The agent was not dietetic as the patient's diet had contained plenty of both antirachitic and antiscorbutic elements. Changes were present at birth in some cases. Fractures were almost certainly due to deficiency of osteoid tissue and absence of lime, bony trabeculæ being replaced by trabeculæ of fibrous tissue, especially near the epiphyses.

Accessory Bone Representing Tubercle of Scaphoid of Foot.—Mr. R. C. ELSMLIE showed a girl, aged 12 years. Pain had been noticed over right scaphoid tubercle twelve months ago after skipping. Talipes valgus right and left. X rays showed accessory bone on right and left side. The right foot was large.

Curious Deformity of Ulna following Injury.—Mr. H. A. T. FAIRBANK showed a boy, aged 13 years, who had fractured his right ulna six years ago. The hand was displaced to the ulnar side, the ulna being very short. In the lower third of the forearm a piece of bone was felt projecting forwards among the flexor muscles. Skiagram: Ulnar shaft much shortened and with a piece of bone nearly 1 in. long projecting forwards from the lower extremity. Epiphysial line oblique; centre for the lower epiphysis of good size but of abnormal shape. On the tip of the piece of bone projecting forwards was a separate piece of bone, apparently a detached portion of epiphysis. The radial epiphysis was set obliquely on the shaft, corresponding with the ulnar displacement of the hand. It was suggested that the condition was the result of splitting of the ulnar epiphysis and shaft, the defoliated portion having grown, while growth at the main portion of the epiphysial line had been deficient. The treatment suggested was removal of the piece of bone projecting forwards and lengthening of the ulna.

Abstracts from Current Literature.

Diseases of Nutrition.

Ætiology of rickets (*'Lancet,'* 1922, I, p. 825).—L. FINDLAY states that rickets is due to confinement, lack of exercise and defective hygiene. Numerous cases among identically fed animals where rickets developed in those not exercised are described. Five hundred rachitic children were compared with 500 normals. There was no real difference in the length of breast-feeding in the two classes, and no striking evidence of gastro-enteritis among the rachitic. Two hundred and sixty of the 500 rachitic children lived in overcrowded quarters, and 340 were taken out-of-doors only very occasionally. Removal of the thymus does not render animals more susceptible, nor does it cause rickets. The average air space per person was in the case of rickets 396 cubic feet, in the case of slight rickets 452, and in the non-rachitic 565. Maternal care was good in 46 per cent. of the rachitic and in 84.5 per cent. of the non-rachitic. Diet played no important part, as the caloric intake in the rachitic was 3315, and in the non-rachitic 3390. Forty-eight per cent. of the rachitic and 40 per cent. of the normals consumed less than 100 grm. of protein per day. The average daily consumption of fat was in the rachitic 87.3 grm., and for the non-rachitic 96.7 grm. Family income did not seem to bear any relation to the disease, the average daily cost per head in the rachitic being 9½d., and in the non-rachitic 10½d. Rickets is not an infective condition. No specific antibody has been found, and puppies injected with blood from children suffering from active rickets do not develop the disease. Flea-bites were found in the rachitic but in very few of the non-rachitic, but rickets may develop in hospitals where vermin do not exist. Hess found that no increased rachitic tendency occurred in children fed on a diet so poor in animal fat as cotton-seed oil, which is totally devoid of the antirachitic vitamin. Further, fat digestion and absorption is good in rickets. Rickets is an unknown disease among the poor Indians, who take little fat of any kind and no animal fat, while among

the rich Hindus, whose children are secluded in dark and airless rooms, the disease is common. It was found that massage and electrical treatment enabled children to walk within four weeks, although the X-ray photographs showed no signs of healing in the bones until the expiration of two to three months. On the other hand, cod-liver oil produced very definite signs of healing on X-ray examination, although the power of walking was not restored till much later. Probably the muscles are affected, perhaps on account of the diminution of creatin content and other degenerative causes. Rickets is not due to deficiency of calcium, as the disease may occur in babies fed on cow's milk, which contains four times more calcium than human milk. In rickets the retention of calcium is erratic and occasionally falls below the normal, but in older children the retention of calcium was above the normal. The calcium content of the blood was also normal.

CHRISTOPHER ROLLESTON.

Ætiology of rickets in infants (*Lancet*, 1922, II, p. 7).—**H. Chick** and others investigated the question as to whether rickets is due to faulty dietary with lack of "A" vitamin or to other unfavourable environmental conditions. The influence of diet under constant conditions of hygiene was first investigated and then the influence of light. The infants were placed on either diet 1 or diet 2. Diet 1 consisted of Vienna undiluted fresh milk from stall-fed cows, with addition of 8 to 10 per cent. of sugar. Cereals were included after the age of 5 months, and later on fresh fruit and vegetables. Diet 2 consisted of a standardised full-cream dried milk yielding a fluid containing 13 per cent. solids, protein 3·4 per cent., fat 3·4 per cent., and sugar 5·3 per cent. For infants under 3 months it was more diluted and a small amount of sugar was added. Cod-liver oil was given to all the infants of this group. To both diets 5 to 10 c.cm. of fruit or vegetable juice was added. Infants on diet 1 received 20 per cent. less milk than those on diet 2, and twice the amount of sugar. In diet 1, 50 per cent. of the calories were given in the form of sugar, 10 to 15 per cent. as protein, and 20 to 30 per cent. as fat. In diet 2, sugar accounted for 30 per cent., protein for 20 per cent., and fat for 50 per cent. Sixty-four cases were under observation for 5 to 15 months. Their ages varied from 1 week to 5 months. All were free from rickets on admission as judged on clinical and X-ray findings. The clinical diagnosis was based on the bony stigmata confirmed by X rays. During the summer no case of rickets was detected among 40 infants on both types of diet. In the winter series of 51, 14 developed rickets in the spring, all of whom were on diet 1, and all, with 3 exceptions, were under 6 months old. Craniotabes occurred in 10 of the 14. Enlargement of the spleen was not found in cases of early rickets. It is concluded that rickets has a marked winter incidence, and that protection in winter can be afforded by diet. The methods of treatment were (1) by cod-liver oil, (2) exposure to mercury vapour quartz lamp, (3) outdoor treatment in sun or shade. Thirty-two children were observed, 14 of whom developed the disease in hospital. Their ages varied from 4 to 18 months, but one was aged 2 years. All were on diet 1, except the last-mentioned case. Six were treated by a 50 per cent. emulsion of unrefined cod-liver oil. The initial dose was 1 to 2 grm., reaching the maximum dose of 5 to 10 grm. in 10 days. Healing, demonstrated radiographically by the deposit of calcium in unossified tissue of the end of the long bones, was noted in 2 to 4 weeks from the beginning of treatment. Seven were treated by exposure to the mercury vapour quartz lamp. Three to four exposures of 5 to 30 minutes

were given weekly. Healing began as early, and was as satisfactory as with cod-liver oil. Seven cases were exposed to direct sunlight, 2 were shaded from direct rays of the sun, 3 others were treated by cod-liver oil as well as sunlight. The rate of healing was most rapid in the last group and in the others was directly proportional to the amount of exposure. In control cases removal to the good sanitary conditions of the hospital was not sufficient to bring about any alteration in the rachitic condition. Fresh air and exercise apart from the greater incidence of sunlight do not prevent the onset of rickets, and with a minimal insolation, diet is the controlling factor. There may be a special photo-synthesis of the A vitamin under the influence of sun radiation.

CHRISTOPHER ROLLESTON.

Rickets: The part played by unhygienic social conditions in predisposing to the disease (*Glas. Med. Journ.*, 1922, I, p. 129).—D. Noël Paton gives an account of an investigation into the condition of some families typical of the general condition of the crofter population of the Lewis in the Outer Hebrides, with special reference to dietetic and environmental factors. He considers that the condition of these people not only as regards diet but also as regards housing and mode of life is one calculated to increase their resistance to the onset of rickets. From his study of rickets he comes to the conclusion that it is highly improbable that the disease can be directly caused either by unhygienic surroundings or by defective feeding. The close association of rickets with the conditions of slum life, and the way in which it is apt to affect more than one member of the same family, suggest the possibility of an infective element in its causation, but the infection need not necessarily be a specific one. The onset of xerophthalmia as an accompaniment of arrested growth in the absence of the fat-soluble A factor is an illustration of this predisposition to infection. The fact that cod-liver oil plays at least some part in the prophylaxis and in the cure of rickets is no argument that it does so in virtue of its containing some factor in the absence of which rickets will develop. It is now considered a specific in rickets and its action is probably an indirect one upon the metabolism, so that resistance to the onset of disease is increased, or the morbid process, if established, is overcome. If the action of unhygienic conditions and of defective diet as simply predisposing to the onset of rickets is accepted, the practical question which has to be faced in each place in which the children are largely affected with rickets is whether the preponderant factor is the former or the latter.

J. ALLAN.

Rickets (*Practitioner*, 1922, CIX, p. 36).—H. Thursfield sums up Mellanby's work by saying that animal fats possess anti-rachitic powers to a considerable degree while the vegetable oils have little or none. The most favourable diet for the production of rickets is one containing an adequate amount of protein, an excess of carbohydrate, a sufficiency of water, soluble B and C vitamins, and very little fat. Mellanby attributed the lack of growth in rats and the occurrence of rickets in puppies to absence or relative deficiency of the fat-soluble A vitamin. But to produce rickets growth must occur; further, the addition of lean meat, which possesses little or no fat-soluble A, prevents the disease; and lastly, green vegetables, containing much of that substance, fail to prevent the onset of the malady. Mellanby's final conclusions were that the following conditions tend to prevent rickets: (1) Plenty of calcium and phosphorus; (2) something in fats associated with or

identical with the fat-soluble vitamin; (3) meat and exercise. Rickets are favoured by the opposites of 1, 2 and 3, excess of bread, and excess of the protein moiety of caseinogen free from calcium. McCollum's experiments showed that rats fed on diet deficient in fat-soluble A but with plenty of phosphorus did not develop rickety changes in the bones. A deficiency of fat-soluble A is not the sole cause of rickets, but the level of phosphate in the blood is partly controlled by the amount of fat-soluble "A" vitamin. The conclusions based by Thursfield on Hutchison's Indian work are that she has not proved that the richer Indians really do consume much fat-soluble A, and that she makes no statements as regards the proportions of the disease at different ages of childhood. He further points out that the experimentalists all rely on changes in the long bones as evidences of rickets whereas they are only late manifestations. Rickets is not a pure deficiency disease.

CHRISTOPHER ROLLESTON.

Rickets: A theory of metabolic disturbances associated with tetany (*Brit. Med. Journ.*, 1922, I, p. 379).—D. Noël Paton states that tetany was found in 43 per cent. of the markedly rachitic and in 41 per cent. of the slight cases of rickets. A limitation of the phosphates of the food in proportion to the calcium combined with a diminished supply of fat-soluble "A" vitamin produced in rats a condition resembling rickets. Not all the calcium of the blood is combined with phosphorus as calcium phosphate. Perhaps it may be combined with fats, as phosphorised fats are found in abundance in the liver of the two-weeks-old chick. During the third week cholesterol esters of fatty acids take the place of phosphorised fats. The phosphorus of the blood is arranged as (1) phospholipin, (2) inorganic phosphorus, and (3) nucleic acid. Lecithin is a phospholipin, and in composition is glycerol combined with a fatty acid radicle, phosphoric acid, and cholin. From it are derived the phosphates of the bones, and it is suggested that some modification of the metabolism of lecithin causing the transference of phosphoric acid to bone in an unsuitable form is the cause of rickets. Further, the cholin part of lecithin is excreted as methyl-guanidin, which causes symptoms of tetany which so often occur in rickets.

CHRISTOPHER ROLLESTON.

Fat-soluble deficiency and rickets (*La Pediatria*, 1922, xxx, p. 97).—O. Cozzolino discusses the question whether there is any relation between rickets and a deficiency of fat-soluble vitamins. If the efficacy of cod-liver oil in rickets is indisputable, it is equally indisputable that its curative effects are not comparable in rapidity of action to those which a corresponding vitamin diet exercises on the cure of another avitaminic disease such as infantile scurvy. Even serious rickets has a tendency to spontaneous cure, and this occurs without any change of diet; scurvy, on the other hand, ends inevitably in death if not cured in time by a vitamin diet. If cod-liver oil acts on rickets in virtue of its fat-soluble vitamin, how is it that phosphorus alone gives the same result? A wet-nurse whose infant has developed rickets may suckle another rachitic infant whose rickets may disappear, or another infant which does not become rickety. The author does not agree with the avitaminic theory of rickets, and brings forward many arguments against it.

VINCENT DICKINSON.

Prevalence of rickets in an agricultural county (*Med. Officer*, 1922, I, p. 147).—W. T. G. Boul investigated 100 cases of undoubted rickets,

In 69 of these enlargement and protrusion of the frontal bones was noticed. The heads of the rachitic were slightly larger than the normal: 52 per cent. of the rickety had heads of 22 inches as compared with 46 per cent. of normal children. Fifty-eight per cent. showed Harrison's sulcus and a similar number were pigeon-chested. Fifty-six per cent. had deformities of the extremities, chiefly of the tibiae. Twenty-seven per cent. were stunted in height. Twenty-seven per cent. of the children suffered from bronchitis, and this complication was most common in those with chest deformity. In 26 per cent. adenoids were present. Rickets was most common in fairly large families. Thirty per cent. gave a history of rickets in other members of the family. Fourteen per cent. of the mothers suffered severely from anæmia and debility. Sixty per cent. of children had been entirely bottle-fed, 24 per cent. partly on the bottle and partly on the breast, and only 16 per cent. entirely on the breast. In the bottle-fed class, cow's milk, Robinson's patent groats or barley, Nestlé's milk, glaxo and malted milk had been given. In the partially bottle-fed breast-feeding had been discontinued between the fourth and fifth months. In the breast-fed class none had been weaned under 1 year of age, 5 had not been weaned till 15 months, and in 1 breast-feeding had continued for 2 years. The author suggests that weaning at 9 months would have prevented the disease. The eruption of the first teeth was much delayed. In only 25 per cent. had the teeth appeared before the end of the seventh month, and only 14 walked before the end of the first year. Cases were more frequent in the inland villages, as a better dietary can be obtained by the fisher folk.

CHRISTOPHER ROLLESTON.

Rickets in India ('*Glasg. Med. Journ.*,' 1922, 1, p. 145).—**H. S. Hutchison** and **S. J. Murphy** present an interesting inquiry into the occurrence of rickets in Nasik, a town of about 35,000 inhabitants, and situated about 120 miles north-east of Bombay. The disease was very prevalent among the well-to-do classes, whose women live a secluded life and whose dietary is superior to that of the poorer classes. Among the very poor, who live in mere hovels, rickets is conspicuous by its absence. Early and late rickets both occur in that stratum of society which enjoys the better dietary, but in which the seclusion of women and children is adopted. It is also noteworthy that late rickets attacks females only, and the onset of the disease is closely related to the adoption of the life of seclusion. It is therefore concluded that seclusion is an indispensable factor in the causation of early and late rickets.

J. ALLAN.

Pancreatic disorder in rickets ('*Brit. Med. Journ.*,' 1922, 1, p. 511).—**E. C. Dodds** reports that as a result of the examination of cases of acute rickets in children, distinct evidence was found of pancreatic disorder, as determined by an increase (1) in urinary diastase and (2) in faecal fat content. The diastatic power of the urine is greatly increased in rickets, the mean of 17 cases being 154; it falls to normal during convalescence. The fat in the faeces is increased in rickets, the mean being 75 per cent., as compared with 20 per cent. for a series of non-rickety children. It is therefore suggested that there is a pancreatic lesion in rickets, and a possible explanation of its bearing on the disease is given, namely, that there is a poor production of fatty acids, and consequently a poor absorption of

calcium. Attention is called to the acidosis of rickets. A new line of treatment is suggested, namely, the administration of pancreatic extract containing lipase.
J. ALLAN.

Influence of light in prevention and cure of rickets ('*Lancet*,' 1922, II, p. 367).—A. F. Hess.—Diet is an important factor. Some of the conflicting results depend upon the difficulty of interpreting correctly the pathological conditions of experimental animals. In rats deprived of the A soluble vitamin, osteoporosis rather than rickets results. Rickets has a well-marked seasonal incidence, the maximum number of cases occurring at the end of March. If infants are placed in the sunlight for 30 minutes daily the rachitic lesions of the epiphyses rapidly undergo calcification. The seasonal incidence depends, therefore, upon the variation of sunlight. Rats fed on the standard rickets-producing dietary but exposed to sunlight, the mercury quartz lamp or the carbon arc lamp do not develop rickets. The other factors concerned are the rapidity of growth and the diet. If the latter be increased and adequate growth obtained light treatment may fail to prevent rickets. Pigmentation of the skin which renders the rays inert is another point to be considered. Black rats fed on the rachitic diet and treated by light develop the disease, while the white rodents escape. Neither adequacy of ventilation or degree of temperature are causative factors. The ultra-violet rays are those which protect against the disease, and when filters are employed which cut off all the rays shorter than 475μ , rickets always ensues. The protective rays can pass through garments made of cotton, but not of heavier material. Röntgen rays failed to protect rats against the malady. Such rays do not produce rickets, but osteoporosis—an extreme exhaustion of the marrow. The rays produce an increase in the amount of inorganic phosphorus in the blood, and this amount is found to be least in March and most in the summer, when the incidence of rickets is least. As breast-milk fails to protect against the malady, diet cannot be the dominant or controlling ætiological element. Rickets occurs in about half of the breast-fed babies among the poor, even if the mothers have a liberal and varied diet.
CHRISTOPHER ROLLESTON.

Quartz lamp treatment of rickets ('*Wien. klin. Woch.*,' 1921, xxxiv, p. 241).—P. Erlacher states that the effect of quartz lamp treatment on rickets was first systematically studied with the help of X-ray findings by Huldshinsky, who succeeded in curing all degrees of rickets in children aged from $1\frac{1}{2}$ to $6\frac{1}{2}$ years with the ultra-violet rays in twenty-two to twenty-six sittings. His observations were confirmed by Putzig and Riedel, and recently Erlacher himself has treated forty-two cases of rickets by this method. The cases included early, well developed, severe and chronic examples of the disease in children aged from 1 to 7 years, and every month comparative skiagrams of the epiphyses of the right forearm were taken. At first the treatment was applied every other day, and later daily, beginning with the abdomen and back, which were irradiated for five minutes each at a time. The duration of the treatment was increased by two minutes until it amounted to fifteen minutes each for the abdomen and back. No bad effects were observed. The children were treated either as out-patients or admitted to hospital without any change being made in their diet. No drugs were given. After four weeks' treatment skiagrams showed an increased deposit of calcium in the osteoid tissue. Clinical improvement generally

occurred after six weeks' treatment, spontaneous fractures rapidly united, and osteotomies and osteoclasias became consolidated with a firm callus within four to six weeks. Erlacher concludes that the quartz lamp is a cheap, convenient and effective method of treating rickets. Within a year a single lamp can cure over 1000 patients.

J. D. ROLLESTON.

Ultra-violet radiation in rickets ('*Amer. Journ. Dis. Child.*,' 1922, xxiv, p. 20).—B. Kramer, H. Casparis and J. Howland exposed 5 children, 4 of whom were coloured and 1 white, to the ultra-violet rays of a mercury quartz lamp placed at a distance of 50 cm. from the patient daily for periods varying from 5 minutes at the beginning to 20 minutes at the end of the course. Rickets had been found to exist previous to treatment by radiographic evidence. The inorganic phosphorus concentration of the serum was low at the commencement of treatment—2.7 to 3.2 mgrm.—but increased at the termination of the course to the normal of 6 mgrm. The pigmented skin of the negro did not interfere with the action of the rays.

CHRISTOPHER ROLLESTON.

Rickets, marasmus and scurvy ('*Lancet*,' 1922, II, p. 551).—H. Thursfield points out that recent experimental work on rickets has not improved the clinician's treatment. Fresh air, good sanitation, sunlight, exercise and cod-liver oil have been the sheet-anchor for 25 years. Further deficiency of fat and excess of carbohydrate are the prime causative factors from the standpoint of the hospital physician. The experimentalist judges of the existence of rickets by the bony deformities in the long bones and ribs, which are a late manifestation of the disease. The clinician diagnoses the ailment by the earlier symptoms of (1) head-sweating and restless sleep, (2) loss of muscle-tone, (3) polyuria, (4) pallor, (5) alteration in the bones of the skull. A normal infant does not sweat. If beads of perspiration appear about the face and neck of an otherwise normal infant rickets can be safely diagnosed. Polyuria may replace sweating. The vitamin hypothesis is so far not proved. Both the dietetic school and the hygienic have right on their side. Scurvy, on the other hand, depends entirely upon the absence of the anti-scorbutic vitamin, and most of the cases have been fed on dried milk. A marasmic infant is one who, although fed on an apparently adequate diet, either remains stationary or loses weight. Some of these infants resemble animals fed on a diet insufficient in regard to its "B" vitamin content, but administration of a diet rich in "B" vitamin does not cure the condition. Some observers have had good results with small doses of thyroid extract.

CHRISTOPHER ROLLESTON.

An epidemic of Barlow's disease ('*La Pediatria*,' 1921, xxix, p. 66).—E. Giorgio describes an outbreak of Barlow's disease which occurred in Venice at the end of 1919 and first half of 1920. All the cases showed the characteristic triad of symptoms, viz. anæmia, characteristic changes in the bones and scorbutic gingivitis, except two in whom the lesions of the gums were absent. In almost all the children the classical symptoms of Barlow's disease were associated with rickets. In one case rickets was combined with the spasmodic diathesis. In all the cases dietetic treatment, which is the most valid test of the correctness of the diagnosis, was successful. Barlow's disease is rare in Italy, not only because up to a few years ago artificial feeding was rare in that country, but because patent

foods were not so popular as at present. All the children in the epidemic described belonged to the working classes, and had been fed for a prolonged period on condensed milk.

J. D. ROLLESTON.

Scurvy rickets (*Bull. et mém. soc. méd. des hôp. de Paris*, 1921, 3^e sér., XLV, p. 288).—**J. Comby** remarks that though infantile scurvy has been familiar to pædiatrists since Barlow's description in 1883, general practitioners are still too apt to overlook it. From an experience of 72 cases he has found that in nine out of ten cases the disease had not been recognised before the child has been brought to him. Since the war the disease has become more frequent for two reasons: (1) Scarcity of fresh milk, and the necessity of employing preserved milk and special infant foods on a large scale; (2) failure on the doctor's part to recognise the symptoms of scurvy and an ignorance of its causes and the means to prevent it. The diagnosis is established by the following points, which should always be borne in mind in the case of a child who has been ailing for some time and has been treated by various methods without success: (1) Artificial feeding for six to ten months with sterilised or condensed milk, or infant foods; (2) pains in the bones followed by loss of power in the lower limbs and cries when they are moved—a sign invariably present; (3) ecchymoses in the gums. This sign, though pathognomonic, is not constant, being absent in 22 per cent. of Comby's cases. Prophylaxis consists in the administration of a small quantity of orange juice, grape juice or lemon juice daily to children who have been fed on sterilised or condensed milk. Treatment consists in substituting boiled fresh milk for preserved milk, and giving one or two teaspoonfuls of orange juice daily.

J. D. ROLLESTON.

Syphilis.

Two cases of extra-genital chancre (*Med. J. Austral.*, 1921, 1, p. 312).—**N. Paul**.—A girl, aged 2½ years, contracted syphilis from an infant brother suffering from congenital syphilis. The boy suffered from marked fissuring of the lips, and the girl from a chancre of the lower lip, doubtless contracted from kissing the brother.

F. R. B. ATKINSON.

Craniotabes and syphilitic rickets (*Paris méd.*, 1921, II, p. 493).—**A. B. Marfan** states that though syphilis may give rise to all forms of rickets, as a rule it produces a special clinical form which is distinguished by the following features: (1) Early onset. It is either congenital or appears in the first three or four months of life. This is the most important characteristic, for all the others result from it. (2) Predominance of rachitic changes in the skull-bones. Craniotabes is first observed, and later abnormal prominence of the frontal and parietal eminences. When rickets is due to another cause, especially tuberculosis and severe and persistent digestive disturbances, the skull is often spared or little affected, and hardly ever shows any craniotabes. (3) Fairly marked anæmia, especially during the first few months of life. (4) Chronic enlargement of the spleen. In the course of the second year or even earlier the last two features tend to disappear, but the cranial stigmata persist for long—sometimes for years and even throughout life. Marfan considers most cases of rickets with well-marked deformity observed after the first year as of syphilitic origin. Rickets due to tuberculosis or digestive disturbance as a rule produces only

a slight deformity, because it commences at a period when the skeleton is much more solid and ossification is less affected. Marfan emphasises the fact that craniotabes is not due to a mere delay in ossification, as maintained by Comby, Lasègue, Wickmann and Lesage, but is the manifestation of early rickets, which in most cases is of syphilitic origin.

J. D. ROLLESTON.

The relation between craniotabes and rickets (*'La Pediatria,'* 1921, xxix, p. 643).—**S. de Stefano** gives a table of 52 cases, comparing the family history, mode of feeding, clinical signs and Wassermann reaction in both mother and child. There was admitted parental syphilis in 8 cases, maternal tuberculosis in 1 case, family rickets in 1 case, premature and still births in 26 cases, *i.e.* 50 per cent.; 28 were breast-fed, 15 mixed fed, 9 artificially fed. The Wassermann reaction was positive either in mother or child in 27 cases, which, together with cases of admitted or clinically evident syphilis, makes a total of 37 cases, or 71 per cent. Therefore, in 29 per cent. syphilitic infection could be excluded. The author considers that facts support the theory that a definite intimate relation exists between craniotabes and rickets—that the former represents the earliest manifestation of the latter. This is also shown in the beneficial effects of early treatment by phosphorus. The large number of cases where syphilitic infection existed bears out Marfan's idea of syphilitic rickets.

VINCENT DICKINSON.

Syphilitic rickets through a wet-nurse (*'La Pediatria,'* 1921, xxix, p. 440).—**L. M. Spolverini** describes the case of a child who, owing to the inability of its mother to nurse it, was put to the breast of a young wet-nurse when 2½ months old. The change was at first followed by considerable improvement in the child's condition, but after two months it began to lose weight, became anæmic, with increase in size of the skull and enlargement of the liver and spleen. The wet-nurse and her own child both gave a positive Wassermann reaction, while in both the parents it was negative. Great improvement followed the administration of calomel.

VINCENT DICKINSON.

The teeth in congenital syphilis (*'Zentralbl. f. inn. Med.,'* 1921, xlii, p. 977).—**P. Kranz**, from ten years' study of dental anomalies in his dental practice, maintains that Hutchinson's teeth are by no means always a typical sign of congenital syphilis, having found them in undoubtedly nonsyphilitic individuals and especially in cretins. They are, moreover, relatively uncommon, and Hutchinson himself regarded them as a sure sign of congenital syphilis only when they formed part of the triad. Kranz also mentions that all disturbances of growth in the period of development, including changes in the teeth and gums, have a common cause, for which local mechanical factors or the action of spirochætes, bacilli or intestinal disturbances are not alone responsible, but are mainly due to disturbances of calcium metabolism which originate from endocrine disorders. Owing to the relatively frequent involvement of the endocrine glands in congenital syphilis dental anomalies are frequently found in these patients, but these anomalies are by no means characteristic of congenital syphilis.

J. D. ROLLESTON.

Gastro-intestinal troubles in the syphilitic infant (*Journ. de Med. de Paris*, 1921, XL, p. 426).—A. Jouin remarks that usually during the first few months the congenital syphilitic infant shows some slight intestinal disturbances and its weight does not increase as much as a healthy infant. Vomiting is frequent, and about the third month a chronic apyrexial diarrhoea. The stools are frequent, pale or yellow, with fragments of undigested casein. This diarrhoea does not respond readily to treatment, and the writer suggests it is of endocrine origin, due to the sclerosing effect of the treponema on the endocrine glands, especially the liver, pancreas and spleen. Besides antisyphilitic treatment by mercury and arsenic compounds, a combination of extracts from the liver, pancreas and spleen may be given.

J. PORTER PARKINSON.

Cardiac and vascular lesions in congenital syphilis (*Zentralbl. f. inn. Med.*, 1921, XLII, p. 601).—L. Hahn, from a study of 150 cases of congenital syphilis, concludes that the great majority of vascular neuroses are due to these causes. In addition to the ordinary signs of vaso-motor instability more serious cases of vascular crises in various regions of the body are included in this category. Congenital syphilis acts as a predisposing cause; all the other factors incriminated are of secondary importance. The site of the disease is either the vessels, the vaso-motor centre, or the endocrine glands closely connected with the blood-pressure. Hahn is of opinion that in the diagnosis of congenital syphilis more importance should be attached to the patient's physiognomy and history than to the Wassermann reaction. The presence of congenital mitral stenosis in 90 per cent. of the cases indicated involvement of the whole cardio-vascular system.

J. D. ROLLESTON.

Syphilis in children of school age with heart disease (*New York State Journ. Med.*, 1921, XXI, p. 176).—B. F. Donaldson, after investigating a limited number of children suffering from cardiac affections, comes to the conclusion that syphilis is not a very great factor in the causation of heart disease in children.

J. ALLAN.

Symmetrical gangrene of the feet in hereditary syphilis (*La Pediatria*, 1921, XXIX, p. 301).—G. Berghinz describes the case of a boy, aged 12 months, admitted to hospital for cachexia and oedema of the lower limbs. All movements were normal and there were no signs of lesion of the central nervous system. The Wassermann reaction was positive. Subsequently the oedema became cyanotic, and a condition of gangrene made its appearance in the lower extremities from the ankle to the tips of the toes. The gangrene, which was at first moist, became dry, and the infant died of marasmus. The autopsy showed normal viscera. There was thrombosis of all the vessels below the popliteal space. The brain was normal. The spinal cord was worm-like and came out of the spinal canal easily. Histological examination by Prof. Bonome showed delayed development, as evidenced by the persistence of a wide gutter in place of the anterior longitudinal sulcus and imperfect formation of the anterior columns and bundle of Türck. The scanty fibres of the white commissure were uneven and often varicose. In the grey matter the cellular nuclei of origin of the roots were composed of a scanty number of cells, the roots being thin and consisting of few fibres. The grey matter itself was more than ordinarily rich in small vessels (congenital syphilis). The meninges were thin and presented marked lymphoid infiltration.

VINCENT DICKINSON.

Epitrochlear adenitis and its relation to congenital syphilis (*'La Pédiatrie,'* 1921, xxix, p. 193).—**S. Fabris** investigated a very large number of children suffering from various maladies up to the age of two years, in whom the presence of epitrochlear adenitis was ascertained. In syphilitic cases it was found in about 33 per cent., in other cases below 11 per cent. He therefore regards it as the sign of a general infective process met with more frequently in hereditary syphilis than in any other affection. If not sufficient of itself to justify a diagnosis of congenital syphilis it should give rise to a suspicion of it, and induce further observation to establish the diagnosis. On the other hand its absence cannot be held to exclude syphilis.

VINCENT DICKINSON.

The ravages of congenital syphilis and how to combat them (*'Glasg. Med. Journ.,'* 1921, II, p. 278).—**L. Findlay** directs attention to the value of ante-natal treatment. The prophylactic method of treatment in the first place stands in marked contrast to the curative method in that it influences all varieties of congenital syphilis, including miscarriages and stillbirths. In the second place, children born after this method of treatment invariably have presented no manifestations of the disease, and in only very rare instances have they developed later a positive Wassermann reaction. In the third place, the ease of the ante-natal method of treatment and the avoidance of the pain and misery so many of these young syphilitics undergo in the course of the curative method are striking and urgent recommendations in its favour. He maintains that the treatment of congenital syphilis is the duty of the family physician, and in his opinion there is no necessity for special venereal clinics, nor for the so-called venereal specialist. He pleads strongly for the inclusion of syphilis among notifiable diseases, and he briefly discusses the influence of syphilis on infantile mortality.

J. ALLAN.

Ante-natal treatment of congenital syphilis (*'Glasg. Med. Journ.,'* 1921, II, p. 270).—**J. G. Greenlees** emphasises its importance. Ante-natal treatment with mercury and potassium iodide is uncertain and unreliable. Far superior results are obtainable by treatment with arsenical compounds and mercury. He quotes 15 cases so treated at the Royal Hospital for Sick Children, Glasgow, in every case with success.

J. ALLAN.

Congenital syphilis after salvarsan treatment of the mother (*'Gaz. hebdomadaire des sciences médicales de Bordeaux,'* 1921, XLII, p. 446).—**R. Dupérier** and **Boisserie-Lacroix** record two cases of congenital syphilis in infants whose mothers had been treated by novarsenobenzol. In the first case treatment had been started in the fourth month and had not been carried out vigorously until the sixth month of pregnancy, 3.75 grm. of novarsenobenzol in all having been given. Treatment by intramuscular injections of biniodide of mercury were then continued until the beginning of the ninth month. In the second case arsenical treatment consisting in 3.15 grm. of arsenobenzol immediately preceding fecundation and during pregnancy all treatment was interrupted. It would be a mistake to regard the arsenical treatment as useless in these two cases, as thanks to it pregnancy was brought to full term in each case, and two apparently healthy children were born, the symptoms developing in the course of the first month.

J. D. ROLLESTON.

The post-natal treatment of congenital syphilis (*Glasg. Med. Journ.*, 1921, II, p. 257).—**G. B. Fleming**, after briefly outlining the methods of treatment by mercury and their results, deals with mercurial treatment combined with treatment by the salvarsan group of substances. In considering the results of treatment the criterion of cure adopted was a negative Wassermann reaction which remained negative. Seventy-four cases are analysed and he thinks the following points are demonstrated: (1) The intravenous injection of these compounds reinforced by mercury is the best post-natal method of treating congenital syphilis. (2) The younger the patient the greater is the likelihood of a cure resulting. (3) Although signs and symptoms disappear and the Wassermann reaction becomes negative immediately after the course of treatment has ceased, there is still a possibility of a positive Wassermann reaction being obtained at a later date, and therefore we should not pronounce the case cured until a negative Wassermann reaction has been obtained more than six months subsequent to the conclusion of treatment.

J. ALLAN.

Some studies in the early treatment of congenital syphilis (*New York Journ. State Med.*, 1922, XXII, p. 127).—**T. B. Givan** maintains that by treating the cases early certain stigmata which often occur in late congenital syphilis will be prevented, as interstitial keratitis, auditory nerve changes, bone changes or cerebro-spinal lues. He outlines the methods of treatment in two series of cases. In the first (15 cases) there was no antenatal treatment; in the second (27 cases) treatment was pre- and post-natal. Mercury internally and externally, combined in certain instances with salvarsan, is advocated. The Wassermann test should be done at intervals.

J. ALLAN.

Neosalvarsan in the treatment of infantile syphilis (*La Pediatria*, 1922, xxx, p. 115).—**R. Spanò** has treated 42 children, aged from 2 months to 10 years, using the drug intravenously in strong solution in twice distilled water. The initial dose did not exceed 1 cgrm. for each kgrm. weight and the maximum 3 cgrm. per kgrm. The author found no difficulty in the technique, there were no bad effects, and 100 per cent. cures.

VINCENT DICKINSON.

Syphilis in children (*Med. Journ. Austral.*, 1922, I, p. 265).—**H. B. Graham**.—Deep subcutaneous injection of novarsenobillon is generally applicable for all syphilitic children. The serious sequelæ are abscesses. Larger doses than usually recommended have produced no unfavourable results. Percutaneous intravenous injections may be preferable in some cases. An analysis of 76 cases is given.

F. R. B. ATKINSON.

Treatment of congenital syphilis by subcutaneous injections of neosalvarsan (*Gaz. hebdom. des sci. méd. de Bordeaux*, 1921, XLII, p. 590).—**R. Lartigaut** states that since 1920 more than 500 subcutaneous injections of neosalvarsan have been given in Rocaz's service to 22 patients at the Children's Hospital at Bordeaux in the treatment of congenital syphilis. The dose consisted of 2 cgrm. of neosalvarsan per kilo of body-weight. Two injections were given a week, and the treatment was stopped after two series of 15 to 20 injections with an interval of a fortnight between each. It is advisable to continue the treatment until the Wassermann reaction in the

blood becomes negative. No bad effects were observed in any case. Local reactions were exceptional and always slight, and a general reaction never occurred. The method proved to be simple, efficacious and safe.

J. D. ROLLESTON.

Sulfarsénol in congenital syphilis (*Glasg. Med. Journ.*, 1921, II, p. 263).—E. Crawford reports on a series of 35 cases submitted to treatment with sulfarsénol. The results, however, showed that the intramuscular injection of this medicament is not as efficacious in producing a cure of congenital syphilis as the intravenous injections of kharsivan—at least when tested by the behaviour of the Wassermann reaction.

J. ALLAN.

Intravenous injection of cyanide of mercury in the infant (*Paris Méd.*, 1921, II, p. 374).—G. Blechmann states that Abadie in 1890 was the first to treat severe lesions of the fundus by intravenous injections of cyanide of mercury, and that Renault in 1902 made use of the method in the syphilis of adults and children. Blechmann has treated several infants suffering from congenital syphilis by this method, the injection being made into the veins of the scalp. The average dose was 1 mgrm. of the drug. Intolerance is indicated by salivation, excess of mucus in the stools, restlessness, insomnia and extreme thirst.

J. D. ROLLESTON.

Treatment of Parrot's disease (*Gaz. hebd. des sci. méd. de Bordeaux*, 1922, XLIII, p. 83).—R. Dupérié and Encontre report their observations on 7 infants with Parrot's pseudo-paralysis who were given the following treatment: (1) Daily inunctions with mercury ointment; (2) 1 mgrm. soon followed by 2 mgrm. of perchloride of mercury by mouth in the form of van Swieten's fluid; (3) after two or three days' mercurial treatment 2 subcutaneous injections of sulfarsénol were given, first in doses of $\frac{1}{2}$ cgrm., which were soon increased to 2 cgrm. per kilo body-weight a week. After 10 or 20 injections the sulfarsénol was stopped and the mercury resumed. Another series of sulfarsénol injections was given after two months' rest, and then a third series after another interval of two months. The mercury, which was stopped during the injections, was resumed during the intervals. After the sixth or seventh injection of sulfarsénol the pseudo-paralysis disappeared as well as the syphilitic manifestations in the skin and mucous membranes and œdema. The coryza was more persistent and did not disappear until after the tenth injection. Enlargement of the epiphyses subsided after the second series of injections. Improvement of the general condition and anæmia took place rapidly. Visceral symptoms and delay in dentition required prolonged treatment.

J. D. ROLLESTON.

Reviews.

THE PHYSIOLOGICAL FEEDING OF INFANTS AND CHILDREN: A HANDBOOK OF THE PRINCIPLES AND PRACTICE OF FEEDING. By ERIC PRITCHARD, M.A., M.D.Oxon., M.R.C.P.Lond., etc. Fourth edition. Pp. 500. London: Henry Kimpton, 1922. Price 21s. net.

THE book is well known to most pædiatricians. The present edition differs from the third in that during the twelve years' interval that has elapsed

between the two editions the author has modified his views with regard to several matters. He now holds the view that babies from birth till six months old should be fed three-hourly, and that it is unphysiological to increase the protein concentration of milk mixtures with increase of age of the infant. We believe these views to be sound, based as they are on experimental and biochemical grounds. The author has also included a chapter on the feeding of children between 1 and 16 years.

The book, as is well known, has as its *leitmotiv* the two principles of "caloric value" and "balance." We believe that the author attaches too much importance to the question of balance (*i. e.* the relative proportions of protein carbohydrate and fat) in the diet of older children, since we do not think that there is as yet any real agreement amongst physiologists as to what constitutes "balance" at any particular age. We fail to be convinced regarding the propriety of continuing to take breast-milk as the standard for children approaching the age of puberty. On the vexed question of raw *v.* cooked milk the author does not write convincingly. He points out that putrefactive organisms in milk "survive long exposure to 100° C.," but a few lines lower down he is "quite convinced that milk maintained at a temperature of 100° C. for three minutes is perfectly safe for infants." Also on the question of starch foods for babies Dr. Pritchard is not quite consistent. On p. 33 and elsewhere he recommends on physiological grounds the giving of minute quantities of starch from the tenth day onwards, and yet on p. 27 he gives bacteriological reasons for regarding starch as a poison to an infant. In addition to these inconsistencies the book contains a number of inaccurate statements, some of which are obviously oversights (as, *e. g.*, the statement on p. 7 that the caloric value of fat is 9.1 *per ounce*). His definitions of food and of basal metabolism are incomplete. The efficiency of the human body is 21 per cent. and not 16 per cent.; and a low solid excretion in the urine is, in our opinion, no evidence of deficiency of food. There are also a number of misprints—such as *stearopsin*, *Meckel's diverticulum*, etc., and above all the author is much too fond of frequently repeating himself.

Notwithstanding these minor faults the book as a whole is a good one and will be found useful by medical officers of children's institutions.

W. M. F.

THE DISEASES OF CHILDREN: MEDICAL AND SURGICAL. By the late HENRY ASHBY, M.D., F.R.C.P., and the late G. A. WRIGHT, M.B., F.R.C.S. Revised by HUGH T. ASHBY, M.D., M.R.C.P. Lond., Hon. Physician to Manchester Children's Hospital; and CHARLES ROBERTS, M.B., F.R.C.S., Hon. Consulting Surgeon to the Manchester Children's Hospital. Sixth edition. Pp. 769. Oxford Medical Publications. London: Henry Frowde & Hodder & Stoughton, 1922. Price £2 2s.

In extending a welcome to the sixth edition of this well-known text-book, the reviewer is constrained to think of the many difficulties confronting those responsible for its re-issue in revised form. It is no easy task to revise a text-book, and the difficulties must be increased where two revisers are at work upon a book originally the result of a collaboration. The ideal to be attained, we presume, is the production of a work which has a uniform appearance of having been recently written, while it profits by the experience and teaching of former writers. Judged by this standard we cannot say that we think that the present revisers have been entirely successful. The book shows evidences of different hands and different ages; the unities,

as it were, have not been preserved. In particular the antique woodcuts should certainly be entirely omitted, and the more modern illustrations, which are really excellent, should be multiplied. We think that the revisers would have done well to have permitted themselves greater freedom in their work.

We offer this criticism because we are, and always have been, attached to the book. For one thing it has adhered to the difficult, but probably correct plan, of combining both medical and surgical diseases of children in one volume. The descriptions of the various diseases are clear, concise and easy to grasp, and although some curious omissions might be noted, the work as a whole is one of which the Manchester School may justly be proud.

R. M.

INFANT FEEDING. By CLIFFORD G. GRULEE, A.M., M.D., LL.D. Fourth edition. Pp. 397. Philadelphia and London: W. B. Saunders Co., 1922. Price 22s. 6d. net.

THE fourth edition of Dr. Grulee's work on infant feeding maintains its previous high standard, and is sufficient indication that it is widely appreciated. Nearly one-third of the text is devoted to the fundamental principles of infants' nutrition, including a valuable chapter on the bacteriology of the gastro-intestinal tract, and another on the observations of various workers on absorption and metabolism. The directions for the management of breast-feeding and artificial feeding are mainly on the same lines as those practised in this country. The author strongly advocates four-hourly feeds, weaning at nine months, and malt sugars in preference to cane or milk sugar. Apparently he thinks more frequent feeds rarely, if ever, necessary, and he believes in the dilution of cow's milk and simple measures generally. The milk of other animals, various modifications of cow's milk and dried milks receive sufficient consideration, but more might have been said about some of the proprietary foods. Barely one page is spent on nourishment during the second year, and the diet recommended consists of milk and carbohydrates, with some vegetable or fruit juices, up to eighteen months of age. Eggs, fish, chicken and red meat are not advised until this age is reached, whereas in this country such foods are generally given with advantage at an earlier age. Grulee appears greatly influenced by German authorities, and he adopts in a slightly modified form Finkelstein's classification of nutritional disturbances into those of weight disturbance, dyspepsia, decomposition (marasmus, etc.) and intoxication (summer diarrhoea, etc.), in spite of the æsthetic objection to the use of the two last-named terms in conveying to a fond mother the nature of the child's illness. Separate chapters are devoted to feeding in premature children, the exudative diathesis, the spasmophilic diathesis, the nervous infant, rickets, scurvy, eczema, pyloric stenosis and in other diseases. These are sufficiently full and practical, and the vitamins have received the amount of attention they deserve at present. The book is well printed in clear type on unglazed paper and has a good index. It is written in excellent language, free from expressions which sometimes in American works jar on British ears. Some of the eight coloured plates, seven being of infant stools which do not convey as accurate a picture as the real article, and of the twenty-nine other illustrations might be omitted and thereby reduce the price of the book. It is such a valuable, scientific and practical work on an important and difficult subject that it deserves a wide circulation, and should certainly be in the possession of all interested in this subject.

E. C.

THE SURGICAL DISEASES OF CHILDREN. By FREDERICK C. PYBUS, M.S., F.R.C.S. London: H. K. Lewis & Co., Ltd., 1922. Price 18s.

It is with very great pleasure that we sit down to review this work. We can state at once it is an excellent book, and any criticisms we may make are merely due to the ordinary disagreement which must and should exist between friends. This book is the result of the author's own practical experience, teaching and writings, and is redundant with the author's ideas.

In surgical books on children it is extremely difficult not to encroach on diseases of adults, and to confine oneself merely to the affections of children. This book has succeeded admirably in dealing solely with the conditions of children, and with these it has dealt very clearly within the limited space at its disposal.

From the practical side the author has wisely ignored all but the outlines of pathology, which, to the practitioner, is of very little value, but we should have been very much better pleased if in the ætiology he had used figures and percentages rather than the words "rare," "unusual" and "common."

The author makes no reference to the works and ideas of others, though he has absorbed these to good purpose and hands them on to his readers. This is carried rather far in some cases, as in dealing with cleft palate he makes no mention of the Lane and Brophy operations, not even to condemn them.

The illustrations are very good, very clear and very plentiful, and reveal the care and interest the author has always taken in his subject. If one might say so, they are infinitely superior to the diagrams he sometimes uses to illustrate his meaning.

There are several statements with which one does not altogether agree. One is very surprised, for instance, to find a writer on children's diseases still making the statement that "all inguinal hernias in childhood are due to a congenital defect in the persistence of a peritoneal process." Can this be proved? A few are congenital—continuation of the tunica with the peritoneum shows this—but surely the majority are acquired by the raising of intra-abdominal pressure? The commonest hernia is the umbilical; these must all be acquired in this way. We really thought that such ideas had disappeared when those who wrote on the diseases of children ceased to regard them as adults in miniature. Still more astonished are we to find that, in dealing with retro-pharyngeal abscess, the tuberculous pre-vertebral abscess is still confounded with this acute form of suppuration, with which pathologically, practically, and from the point of view of treatment it has nothing whatever in common.

In the treatment of psoas abscess we note that the author still injects iodoform, which we should have thought everybody now at last recognised to be a perfectly inert substance; its claim as a disinfectant has long been discarded.

Curiously enough in dealing with intussusception, though no percentages are given, Mr. Pybus gives the ileo-colic intussusception as the commonest of types—he must surely be alone in such an opinion. In the diagrams which accompany this form of intussusception it is quite rightly labelled "enteric"; its name is changed to ileo-colic on its reaching the ileo-cæcal valve. One would like to know how far an enteric intussusception must start above the valve in order to maintain its proper name. We thought that there had been a general movement to discard fanciful and wholly unnecessary names, and to class intussusceptions as enteric, ileo-cæcal and colic. We have great pleasure, however, in noting that the author admits that the

apex of the intussusception remains constant throughout its formation. In the old explanation of the ileo-colic variety the possibility of its existence depended solely on the fact that the apex was constantly changing.

We are rather doubtful of the statement that "the tonsils are frequently tuberculous." We thought 5 per cent. of tuberculous tonsils was about the most claimed by investigators, and that it was recognised that the injury done to the glands of the neck was rather by the absorption of sepsis from the teeth, tonsils and adenoids, preparing a suitable nidus in which the circulating tubercle bacilli were able to settle.

If Mr. Pybus can forgive these little criticisms upon quite minor points in an otherwise very excellent book we can assure him that we welcome his book entirely, as a great help and useful addition to the surgery of children, and a very great help and assistance to the general practitioners and students, for whom largely it is evidently written. D. C. L. F.

SYPHILIS. Tome II: SYPHILIS ACQUISE DE L'ENFANCE ET SYPHILIS HÉRÉDITAIRE. Par ED. FOURNIER et PIERRE FERNET. Pp. 279. Paris: Maloine et fils, 1921. 68 figures et 8 planches en couleurs. Price 18 frs.

THIS is the twentieth volume of the 'Traité de Pathologie médicale et de Thérapeutique appliquée,' edited by Emile Sergent, L. Ribadeau-Dumas and L. Babonneix. In the section on acquired syphilis in infancy, Fernet discusses the possibility of infection of the child during birth from lesions on the mother's genital organs, assuming that, however late in pregnancy the mother is infected, the child is always syphilised. He points out the importance of discovering a chancre, usually cephalic, in the diagnosis of acquired syphilis in infants, but mentions that it is often not found, and may be of short duration. He states that syphilis acquired by infants may be followed by dystrophic effects similar to those of heredo-syphilis, and quotes a case reported by Eudlitz of an infant which contracted syphilis from a syphilitic nurse, and at the age of thirteen had keratitis, malformed teeth, and almost complete deafness and arrested sexual development.

The second and largest section of the book, by Edmond Fournier, gives a full and lucid description of early congenital syphilis and is well illustrated. Fournier points out that heredo-syphilis may cause latent visceral lesions which may lay the foundation for visceral disease in later life. The subject of the transmission of syphilis to the infant is dealt with only briefly, and the question of paternal heredity is left open. Fournier gives at length the evidence for transmission of heredo-syphilis to the second generation (syphilis of the third generation), including his own observations, and concludes that apart from the transmission of dystrophic effects, actual syphilitic lesions occur in 14 per cent. of such cases. He quotes a case of apparent transmission to the fourth generation (Gaucher). Fournier holds the view that every child born of syphilitic parents should be treated even if apparently healthy. In syphilitic affection of the endocrine glands, which is alleged to be the cause of certain dystrophic effects, such as hypothyroidism in the case of the thyroid, and infantilism in the case of the genital organs, administration of extract of the corresponding gland is advised in addition to anti-syphilitic treatment. Fournier has strong views on the question of the marriage of syphilitics, and, contrary to the majority of syphilologists, holds that syphilis should be a bar to marriage. He supports this opinion by quoting cases of women who gave birth to syphilitic infants twenty years

after infection, the husband being healthy. Such cases, however, are obviously open to other explanations.

The last section, on late heredo-syphilis, is by Fernet. Discussing the frequent difficulty in diagnosis between heredo-syphilis and tuberculous lesions, he supports the view that syphilis creates a soil which is vulnerable to tubercle. In the same way it predisposes to rickets, which is not, as Parrot taught, syphilitic in nature, but may be of syphilitic origin. With regard to prognosis in late heredo-syphilis, Fernet mentions the observations of Hutinel and Nadal on recrudescence of heredo-syphilis under the influence of acute infections, either in the form of actual syphilitic lesions, or, more often, by the abnormal evolution of the superadded infection. Immunity in heredo-syphilis, he says, disappears between the ages of eighteen and thirty, so that a heredo-syphilitic may contract the acquired disease, but the gravity of the latter is diminished in such cases (Gaucher and others). Reinfections occur chiefly in subjects who have not been severely affected by the inherited disease, and who have a negative Wassermann reaction. In a case reported by Joltrain, however, this reaction was weakly positive.

The book constitutes a concise and comprehensive monograph on syphilis in childhood. C. F. M.

OTO-RHINO-LARYNGOLOGY FOR THE STUDENT AND PRACTITIONER. By Dr. GEORGES LAURENS. Authorised English Translation of the Fourth Revised French Edition by H. CLAYTON FOX, F.R.C.S.Irel. With a foreword contributed by Sir J. DUNDAS GRANT, M.A., M.D., F.R.C.S. The Second English Edition with 589 illustrations. Bristol: John Wright & Sons, 1922. Price 17s. 6d. net.

THE rapid appearance of a second English edition of this excellent textbook so soon after the first (see *BRITISH JOURNAL OF CHILDREN'S DISEASES*, xvii, p. 58) is a convincing proof of the success which it deserves. In the present edition the text has been revised, some chapters have been condensed, and certain additions made, especially on Vincent's angina, the treatment of hay-fever, pseudo-hæmoptysis of laryngeal origin and vaccine therapy.

J. D. R.

MEDICINE: ANALYTICAL REVIEWS OF GENERAL MEDICINE, NEUROLOGY AND PEDIATRICS. Edited by DAVID L. EDSALL, Harvard Medical School, JOHN HOWLAND, Johns Hopkins Medical School; Associate Editor, PAUL D. WHITE, Massachusetts General Hospital. Published quarterly by Williams & Williams Company, Baltimore, U.S.A. Subscription price per volume net, post paid, \$5.00 United States, Mexico, Cuba, \$5.25 Canada, \$5.50 other countries. Single copies \$2.00.

WE welcome the arrival of this new quarterly, which made its first appearance last May. Its function is to supply authoritative and comprehensive reviews of subjects in the field of internal medicine, neurology and pediatrics. The articles in the first number, which admirably fulfil this aim, are by Dr. G. Canby Robinson on "The Therapeutic Use of Digitalis," and by Dr. Kenneth D. Blackfan on "The Treatment of Meningococcus Meningitis."

J. D. R.

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